

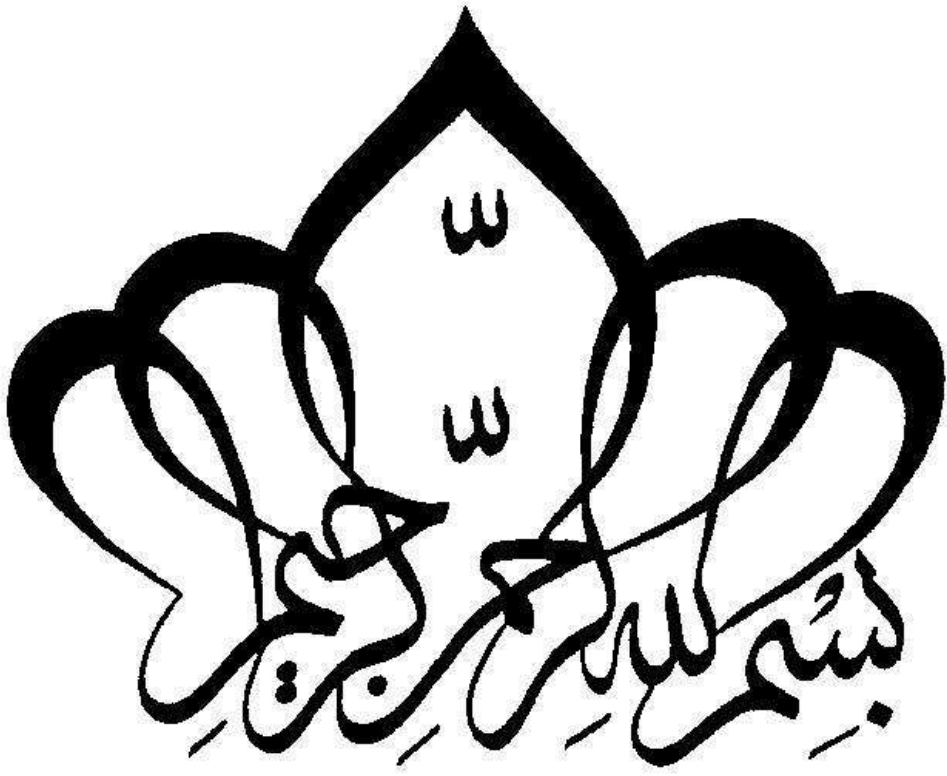
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2600 MCQ with Illustrated Answer
Full Topics Note
14 Chapter

Cardiology

P. M MCQ Bank 2017





قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
۞ إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

إهداء

للكل آية من كتاب الله حفظناها ولكل معلومة قرأناها ونسيناها لعل الله يحمينا من النسيان ويروها إلينا
للكل شخص وعونا له في ظهر الغيب لعل الله أن يستجيب دعائنا
للكل مريض قرأ له ما أله لعل الله يشفيه ويعير إليه صحته وعافيته
للكل لحظة تألم بها أحببنا لعل الله لا يريهم الألم ولا يضييهم مرة أخرى
للكل موقف احتجت به عيناى فلم تلبيانى لعل الله لا يضرنى بهما في حياتي
للكل حالم صاحب أهراء ومباوى لعل الله يحقق له مقاصده حتى يرضيه ويرضى عنه
للكل مجتهد لعل الله يجعل له من التوفيق أوفر حظ ونصيب ويهون له الأسباب كلها من حيث لا يدري من غير ضراء مضرة ولا فتنة مضلة
للكل من فقر شيئا في الحياة لعل الله يعيره إليه أو يعوضه بما يسعده
للكل شخص ابتعدنا عنه لعل الله يجمعه بمن هم أفضل منا
وأخيرا وليس آخرا لأهلنا لعل الله يجعلها لهم صرقة جارية ترو إليهم قليلا من فضلهم علينا وتكون سببا يلم شملنا تحت سقف واحد بعد أن
هجرتنا الحروب والمصائب

يقول أحد الصالحين

إِذَا رَأَيْتَ أَنَّ اللَّهَ وَفَقَكَ لِإِخْوَةِ تَعْيْنِكَ عَلَى الْخَيْرِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ بِكَ خَيْرًا !! وَإِذَا وَفَقَكَ اللَّهَ لِلدُّعَاءِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ أَنْ يُعْطِيَكَ !! وَإِذَا
وَفَقَكَ لِقَضَاءِ حَوَائِجِ النَّاسِ فَأَعْلَمْ أَنَّ لَكَ يَتَخَلَّى عَنْكَ !! وَإِذَا وَفَقَكَ اللَّهَ لِذِكْرِهِ.. فَأَعْلَمْ أَنَّكَ مُحِبٌّ !! .. وَإِذَا أَحْبَبَّكَ أَحَدٌ.. وَنَصَرَكَ.. وَأَيَّرَكَ..
وَأُسْتَجَابَ لِدُعَائِكَ !!

This issue of **MRCPPART1 MADE EASY 2017** contains the following:

- Full topic note.
- Around 2600 MCQs from **PM 2017 BANK** covering 14 chapter in internal medicine
- External links for the related guidelines (such as NICE, SIGN, RCP ...etc).
- Comments with illustrative pictures and links to external media.
- Most Recommended QBank for MRCP Part 1, Arab Board, Jordanian Board and other internal medicine assessment exams.

Please Note that

Each chapter begins with notes section. These notes are repeated under the Questions in the website. In these collections the notes are not repeated under each question except in some to avoid unnecessary repetition. For clarity and convenience the title of the related note is only mentioned after the comments in case the reader want to refer back to the respective note in the notes section.

The illustration below is to show and Example

☐	Minimal change disease
☐	Post-streptococcal glomerulonephritis
☐	Goodpasture's syndrome

Goodpasture's syndrome

- IgG deposits on renal biopsy
- anti-GBM antibodies

• These changes are characteristic of Goodpasture's syndrome.

[Discuss and give feedback](#)

Goodpasture's syndrome

Comment of the Q

title of the note in case the reader want to refer back to the notes sections

MRCPPART1 MADE EASY 2017

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إعداد وتنسيق

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&

A. MURAD

لا تنسونا من صالح دعائكم

ما كان من خطأ فمن أنفسنا وما كان من توفيق فمن الله

تم بحمد الله وتوفيقه ومنه

جعل الله عملنا متقبلاً خالصاً لوجهه الكريم

Reference ranges

Reference ranges vary according to individual labs. All values are for adults unless otherwise stated

Full blood count

Haemoglobin	Men: 13.5-18 g/dl Women: 11.5-16 g/dl
Mean cell volume	82-100 fl
Platelets	150-400 * 10 ⁹ /l
White blood cells	4-11 * 10 ⁹ /l

Urea and electrolytes

Sodium	135-145 mmol/l
Potassium	3.5 - 5.0 mmol/l
Urea	2.0-7 mmol/l
Creatinine	55-120 umol/l
Bicarbonate	22-28 mmol/l
Chloride	95-105 mmol/l

Liver function tests

Bilirubin	3-17 umol/l
Alanine transferase (ALT)	3-40 iu/l
Aspartate transaminase (AST)	3-30 iu/l
Alkaline phosphatase (ALP)	30-100 umol/l
Gamma glutamyl transferase (yGT)	8-60 u/l
Albumin	35-50 g/l
Total protein	60-80 g/l

Other haematology

Erythrocyte sedimentation rate (ESR)	Men: < (age / 2) mm/hr Women: < ((age + 10) / 2) mm/hr
Prothrombin time (PT)	10-14 secs
Activated partial thromboplastin time (APTT)	25-35 secs
Ferritin	20-230 ng/ml
Vitamin B12	200-900 ng/l
Folate	3.0 nmol/l
Reticulocytes	0.5-1.5%
D-Dimer	< 400 ng/ml

Other biochemistry

Calcium	2.1-2.6 mmol/l
Phosphate	0.8-1.4 mmol/l
CRP	< 10 mg/l
Thyroid stimulating hormone (TSH)	0.5-5.5 mu/l
Free thyroxine (T4)	9-18 pmol/l
Total thyroxine (T4)	70-140 nmol/l
Amylase	70-300 u/l

Uric acid	0.18-0.48 mmol/l
-----------	------------------

Arterial blood gases

pH	7.35 - 7.45
pCO2	4.5 - 6.0 kPa
pO2	10 - 14 kPa

Lipids

Desirable lipid values depend on other risk factors for cardiovascular disease, below is just a guide:

Total cholesterol	< 5 mmol/l
Triglycerides	< 2 mmol/l
HDL cholesterol	> 1 mmol/l
LDL cholesterol	< 3 mmol/l

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Acute coronary syndrome: a very basic introduction

♦ **Acute coronary syndrome (ACS)** is an umbrella term covering a number of acute presentations of ischaemic heart disease.

♦ It covers a number of presentations, including:

- ST elevation myocardial infarction (STEMI)
- non-ST elevation myocardial infarction (NSTEMI).
- unstable angina

♦ Before we go into more detail into these presentations it's useful to take a step back and consider how such conditions develop.

♦ ACS generally develops in patients who have ischaemic heart disease, either known or previously undetected. Ischaemic heart disease is a term synonymous with coronary heart disease and coronary artery disease. It describes the gradually build up of fatty plaques within the walls of the coronary arteries. **This leads to two main problems:**

1. Gradual narrowing, resulting in less blood and therefore oxygen reaching the myocardium at times of increased demand. This results in angina, i.e. chest pain due to insufficient oxygen reaching the myocardium during exertion
2. The risk of sudden plaque rupture. The fatty plaques which have built up in the endothelium may rupture leading to sudden occlusion of the artery. This can result in no blood/oxygen reaching the area of myocardium.

♦ **Remember that there are a large number of factors which can increase the chance of a patient developing ischaemic heart disease:**

Unmodifiable risk factors	Modifiable risk factors
Increasing age Male gender Family history	Smoking Diabetes mellitus Hypertension Hypercholesterolaemia Obesity

Pathophysiology

Ischaemic heart disease is a complex process which develops over a number of years. **A number of changes can be seen:**

- initial endothelial dysfunction is triggered by a number of factors such as smoking, hypertension and hyperglycaemia
- this results in a number of changes to the endothelium including pro-inflammatory, pro-oxidant, proliferative and reduced nitric oxide bioavailability
- fatty infiltration of the subendothelial space by low-density lipoprotein (LDL) particles
- monocytes migrate from the blood and differentiate into macrophages. These macrophages then phagocytose oxidized LDL, slowly turning into large 'foam cells'. As these macrophages die the result can further propagate the inflammatory process.
- smooth muscle proliferation and migration from the tunica media into the intima results in formation of a fibrous capsule covering the fatty plaque.

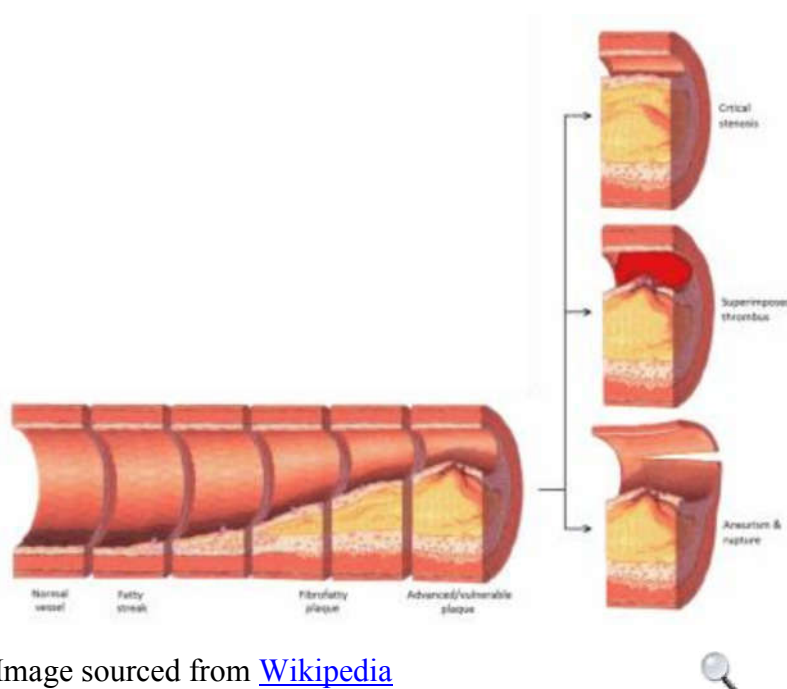


Image sourced from [Wikipedia](#)

Diagram showing the progression of atherosclerosis in the coronary arteries with associated complications on the right.

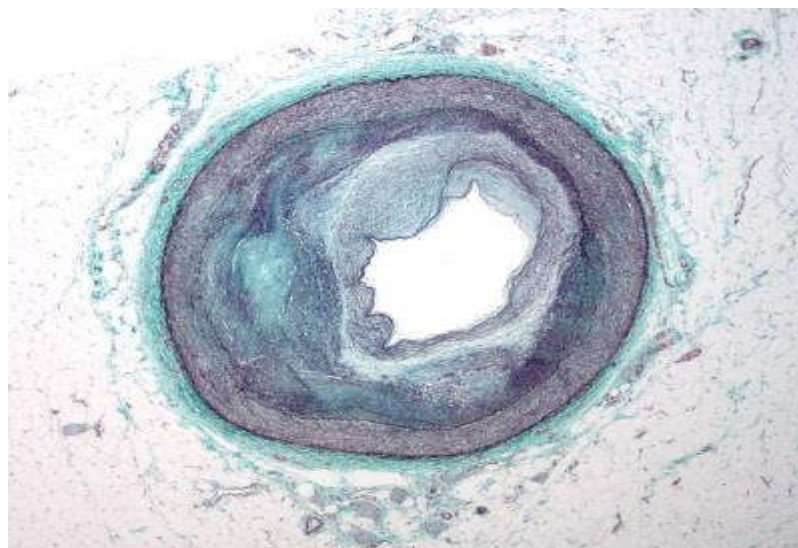


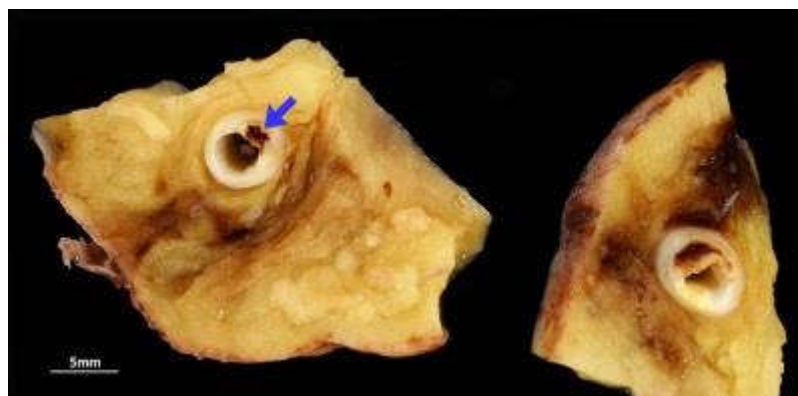
Image sourced from [Wikipedia](#)

Slide showing a markedly narrowed coronary artery secondary to atherosclerosis. Stained with Masson's trichrome.

Complications of atherosclerosis

Once a plaque has formed a number of complications can develop:

- the plaque forms a physical blockage in the lumen of the coronary artery. This may cause reduced blood flow and hence oxygen to the myocardium, particularly at times of increased demand, resulting clinically in angina
- the plaque may rupture, potentially causing a complete occlusion of the coronary artery. This may result in a myocardial infarction



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Ruptured coronary artery plaque resulting in thrombosis and associated myocardial infarction.



© Image used on license from PathoPic

Pathological specimen showing infarction of the anteroseptal and lateral wall of the left ventricle. There is a background of biventricular myocardial hypertrophy.

Symptoms and signs:

The classic and most common feature of ACS is chest pain.

- typically central/left-sided
- may radiate to the jaw or the left arm
- often described as 'heavy' or constricting, 'like an elephant on my chest'
- it should be noted however in real clinical practice patients present with a wide variety of types of chest pain and patients/doctors may confuse ischaemic pain for other causes such as dyspepsia
- certain patients e.g. diabetics/elderly may not experience any chest pain

Other symptoms in ACS include:

- dyspnoea
- sweating
- nausea and vomiting

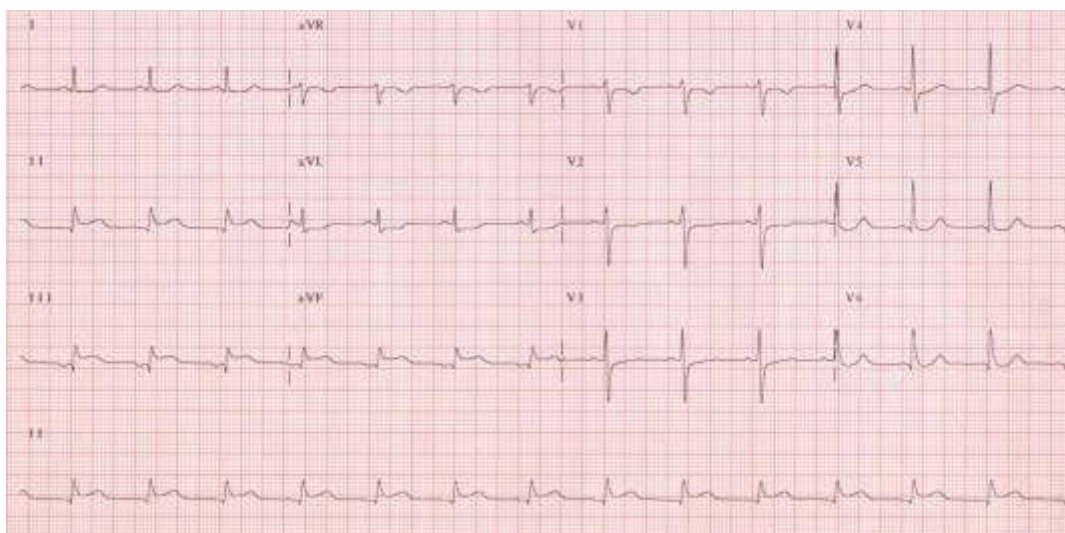
Patients presenting with ACS often have very few physical signs to elicit:

- pulse, blood pressure, temperature and oxygen saturations are often normal or only mildly altered e.g. tachycardia
- if complications of the ACS have developed e.g. cardiac failure then clearly there may a number of findings
- the patient may appear pale and clammy.

Investigations

The two most important investigations when assessing a patient with chest pain are:

- ECG
- cardiac markers e.g. troponin



ECG showing a ST elevation myocardial infarction (STEMI). Note by how looking at which leads are affected (in this case II, III and aVF) we are able to tell which coronary arteries are blocked (the right coronary artery in this case). A blockage of the left anterior descending (LAD) artery would cause elevation of V1-V4, what is often termed an 'anterior' myocardial infarction.



© Image used on license from [Dr Smith, University of Minnesota](#) 🔍 🔍

ECG showing a non-ST elevation myocardial infarction (NSTEMI). On the ECG there is deep ST depression in I-III, aVF, and V3-V6. aVR also has ST elevation. Deep and widespread ST depression is associated with very high mortality because it signifies severe ischemia usually of LAD or left main stem.

The table below shows a simplified correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anterior	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Lateral	I, V5-6	Left circumflex



Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

Management

Once a diagnosis of ACS has been made there are a number of elements to treatment:

- prevent worsening of presentation (i.e. further occlusion of coronary vessel)
- revascularise (i.e. 'unblock') the vessel if occluded (patients presenting with a STEMI)
- treat pain

A commonly taught mnemonic for the treatment of ACS is **MONA**:

- Morphine
- Oxygen
- Nitrates
- Aspirin

Whilst useful it should be remembered that not all patients require oxygen therapy. British Thoracic Society guidelines are now widely adopted and oxygen should only be given if the oxygen saturations are < 94%.

Chapter: cardiology

For patients who've had a **STEMI** (i.e. one of the coronary arteries has become occluded) the priority of management is to reopen, or revascularise, the blocked vessel.

- a second antiplatelet drug should be given in addition to aspirin. Options include clopidogrel, prasugrel and ticagrelor
- for many years the treatment of choice was thrombolysis. This involved the intravenous administration of a thrombolytic or 'clot-busting' drug to breakdown the thrombus blocking the coronary artery
- since the early 2000's thrombolysis has been superseded by percutaneous coronary intervention (PCI). In this procedure the blocked arteries are opened up using a balloon (angioplasty) following which a stent may be deployed to prevent the artery occluding again in the future. This is done via a catheter inserted into either the radial or femoral artery

If a patient presents with an **NSTEMI** then a risk stratification tool (such as GRACE) is used to decide upon further management. If a patient is considered high-risk or is clinically unstable then coronary angiography will be performed during the admission. Lower risk patients may have a coronary angiogram at a later date.

Secondary prevention

Patients who've had an ACS require lifelong drug therapy to help reduce the risk of a further event. **Standard therapy comprises the following as a minimum:**

- aspirin
- a second antiplatelet if appropriate (e.g. clopidogrel)
- a beta-blocker
- an ACE inhibitor
- a statin

Further images

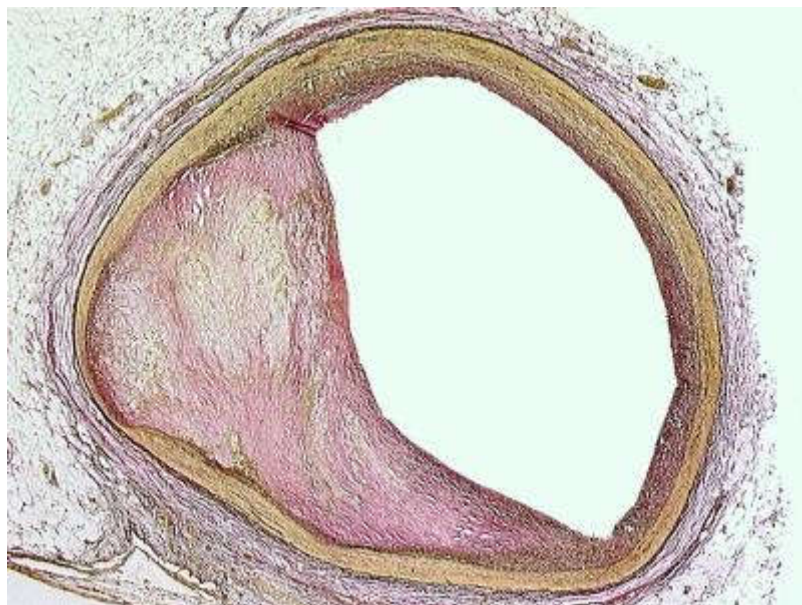
The following images show the progress of coronary artery atherosclerosis:



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Normal coronary artery with blood in the lumen.



© Image used on license from PathoPic

Slightly stenosed coronary artery



© Image used on license from PathoPic

Moderately stenosed coronary artery, between 50-75%



© Image used on license from PathoPic

Severely stenosed coronary artery



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Recanalised old atherothrombotic occlusion of a coronary artery. Numerous small neolumina recanalising the organised occluding thrombus (indicated with arrows).

Acute coronary syndrome: management of NSTEMI

◆NICE produced guidelines in 2013 on the *Secondary prevention in primary and secondary care for patients following a myocardial infarction management of unstable angina and non-ST elevation myocardial infarction (NSTEMI)*. These superceded the 2010 guidelines which advocated a risk-based approach to management which determined whether drugs such as clopidogrel were given.

All patients should receive:

- aspirin 300mg
- nitrates or morphine to relieve chest pain if required

◆Whilst it is common that non-hypoxic patients receive oxygen therapy there is little evidence to support this approach. The 2008 British Thoracic Society oxygen therapy guidelines advise not giving oxygen unless the patient is hypoxic.

Antithrombin treatment. Fondaparinux should be offered to patients who are not at a high risk of bleeding and who are not having angiography within the next 24 hours. If angiography is likely within 24 hours or a patients creatinine is $> 265 \mu\text{mol/l}$ unfractionated heparin should be given.

Clopidogrel 300mg should be given to all patients and continued for 12 months.

Intravenous **glycoprotein IIb/IIIa receptor antagonists** (eptifibatide or tirofiban) should be given to patients who have an intermediate or higher risk of adverse cardiovascular events (predicted 6-month mortality above 3.0%), and who are scheduled to undergo angiography within 96 hours of hospital admission.

Coronary angiography should be considered within 96 hours of first admission to hospital to patients who have a predicted 6-month mortality above 3.0%. It should also be performed as soon as possible in patients who are clinically unstable.

The table below summaries the mechanism of action of drugs commonly used in the management of acute coronary syndrome:

Medication	Mechanism of action
Aspirin	Antiplatelet - inhibits the production of thromboxane A ₂
Clopidogrel	Antiplatelet - inhibits ADP binding to its platelet receptor
Enoxaparin	Activates antithrombin III, which in turn potentiates the inhibition of coagulation factors Xa
Fondaparinux	Activates antithrombin III, which in turn potentiates the inhibition of coagulation factors Xa
Bivalirudin	Reversible direct thrombin inhibitor

Chapter: cardiology

External Link

[NICE](#)

2013 Secondary prevention in primary and secondary care for patients following a myocardial infarction

[SIGN](#)

2016 ACS guidelines

[British Thoracic Society](#)

2008 Emergency oxygen therapy guidelines

[NICE](#)

2010 Unstable angina and NSTEMI guidelines

Acute coronary syndrome: prognostic factors

The 2006 Global Registry of Acute Coronary Events (**GRACE**) study has been used to derive regression models to predict death in hospital and death after discharge in patients with acute coronary syndrome.

Poor prognostic factors:

- age
- development (or history) of heart failure
- peripheral vascular disease
- reduced systolic blood pressure
- Killip class*
- initial serum creatinine concentration
- elevated initial cardiac markers
- cardiac arrest on admission
- ST segment deviation

***Killip class** - system used to stratify risk post myocardial infarction

Killip class	Features	30 day mortality
I	No clinical signs heart failure	6%
II	Lung crackles, S3	17%
III	Frank pulmonary oedema	38%
IV	Cardiogenic shock	81%

Acute pericarditis

Pericarditis is one of the differentials of any patient presenting with chest pain.

Features

- chest pain: may be pleuritic. Is often relieved by sitting forwards
- other symptoms include non-productive cough, dyspnoea and flu-like symptoms
- pericardial rub
- tachypnoea
- tachycardia

Causes

- viral infections (Coxsackie)
- tuberculosis
- uraemia (causes 'fibrinous' pericarditis)
- trauma
- post-myocardial infarction, Dressler's syndrome
- connective tissue disease
- hypothyroidism

ECG changes

- widespread 'saddle-shaped' ST elevation
- PR depression: most specific ECG marker for pericarditis



© Image used on license from [Dr Smith, University of Minnesota](#)

ECG showing pericarditis. Note the widespread nature of the ST elevation and the PR depression

External Link

[Life in the fast lane](#)

[Pericarditis review](#)

Adult advanced life support

The joint European Resuscitation Council and Resuscitation Council (UK) 2010 guidelines do not alter significantly from the 2005 guidelines. Please see the link for more details, below is only a very brief summary of key points / changes.

Major points include:

- ratio of chest compressions to ventilation is 30:2
- chest compressions are now continued while a defibrillator is charged
- during a VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock and then every 3-5 minutes (during alternate cycles of CPR). In the 2005 guidelines, adrenaline was given just before the third shock. Amiodarone 300 mg is also given after the third shock
- atropine is no longer recommended for routine use in asystole or pulseless electrical activity (PEA).
- a single shock for VF/pulseless VT followed by 2 minutes of CPR, rather than a series of 3 shocks followed by 1 minute of CPR
- asystole/pulseless-electrical activity should be treated with 2 minutes of CPR, rather than 3, prior to reassessment of the rhythm
- delivery of drugs via a tracheal tube is no longer recommended
- following successful resuscitation oxygen should be titrated to achieve saturations of 94-98%. This is to address the potential harm caused by hyperoxaemia

External Link

[Resuscitation Council](#)

2015 Advanced Life Support guidelines

[Resuscitation Council](#)

2015 Basic Life Support guidelines

Angina pectoris: drug management

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used. If used in combination with a beta-blocker then use a long-acting dihydropyridine calcium-channel blocker (e.g. modified-release nifedipine). Remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs: a long-acting nitrate, ivabradine, nicorandil or ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- the BNF advises that patients who develop tolerance should take the second dose of isosorbide mononitrate after 8 hours, rather than after 12 hours. This allows blood-nitrate levels to fall for 4 hours and maintains effectiveness
- this effect is not seen in patients who take modified release isosorbide mononitrate

Ivabradine

- a new class of anti-anginal drug which works by reducing the heart rate
- acts on the I_f ('funny') ion current which is highly expressed in the sinoatrial node, reducing cardiac pacemaker activity
- adverse effects: visual effects, particular luminous phenomena, are common. Headache. Bradycardia, due to the mechanism of action, may also be seen
- there is no evidence currently of superiority over existing treatments of stable angina

External Link

[NICE](#)

2011 Stable angina guidelines

[Clinical Knowledge Summaries](#)

Angina guidelines

[SIGN](#)

Management of stable angina

[Royal College of Physicians](#)

2013 Pharmacological treatment of chronic stable angina pectoris

Angiotensin II receptor blockers

Angiotensin II receptor blockers are generally used in situations where patients have not tolerated an ACE inhibitor, usually due to the development of a cough.

Examples

- candesartan
- losartan
- irbesartan

Like ACE inhibitors they should be used with caution in patients with renovascular disease. Side-effects include hypotension and hyperkalaemia.

Mechanism

- block effects of angiotensin II at the AT1 receptor

Evidence base

- shown to reduce progression of renal disease in patients with diabetic nephropathy
- evidence base that losartan reduces CVA and IHD mortality in hypertensive patients

Angiotensin-converting enzyme inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are now the established first-line treatment in younger patients with hypertension and are also extensively used to treat heart failure. They are known to be less effective in treating hypertensive Afro-Caribbean patients. ACE inhibitors are also used to treat diabetic nephropathy and have a role in secondary prevention of ischaemic heart disease.

Mechanism of action:

- inhibit the conversion angiotensin I to angiotensin II

Side-effects:

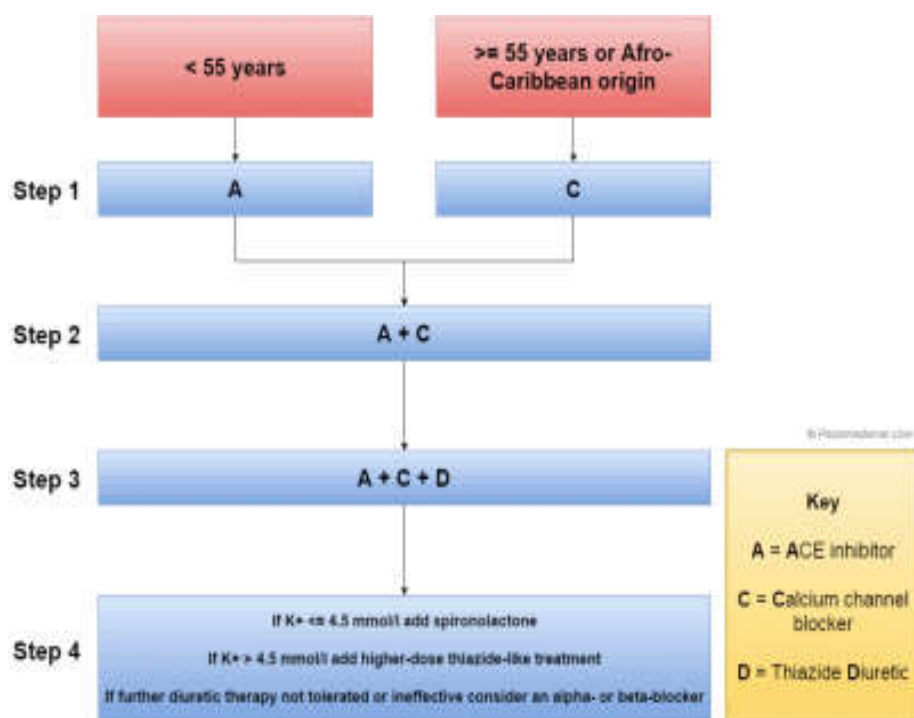
- cough: occurs in around 15% of patients and may occur up to a year after starting treatment. Thought to be due to increased bradykinin levels
- angioedema: may occur up to a year after starting treatment
- hyperkalaemia
- first-dose hypotension: more common in patients taking diuretics

Cautions and contraindications

- pregnancy and breastfeeding - avoid
- renovascular disease - significant renal impairment may occur in patients who have undiagnosed bilateral renal artery stenosis
- aortic stenosis - may result in hypotension
- patients receiving high-dose diuretic therapy (more than 80 mg of furosemide a day) - significantly increases the risk of hypotension
- hereditary of idiopathic angioedema

Monitoring

- urea and electrolytes should be checked before treatment is initiated and after increasing the dose
- a rise in the creatinine and potassium may be expected after starting ACE inhibitors. Acceptable changes are an increase in serum creatinine, up to 30%* from baseline and an increase in potassium up to 5.5 mmol/l*.



Flow chart showing the management of hypertension as per current NICE guidelines

*Renal Association UK, Clinical Knowledge Summaries quote 50% which seems rather high. SIGN advise that the fall in eGFR should be less than 20%. The NICE CKD guidelines suggest that a decrease in eGFR of up to 25% or a rise in creatinine of up to 30% is acceptable

External Link

[Clinical Knowledge Summaries](#)

Managing ACE inhibitors

[NICE](#)

2014 Chronic kidney disease guidelines

Antiplatelet: summary of latest guidance

The table below summaries the most recent guidelines regarding antiplatelets:

Diagnosis	1st line	2nd line
NSTEMI	Aspirin (lifelong) & clopidogrel or ticagrelor (12 months)	If aspirin contraindicated, clopidogrel (lifelong)
STEMI	Aspirin (lifelong) & clopidogrel or ticagrelor (1m if no/bare stent, 12 m if drug-eluting stent)	If aspirin contraindicated, clopidogrel (lifelong)
TIA*	Clopidogrel (lifelong)	Aspirin (lifelong) & dipyridamole (lifelong)
Ischaemic stroke	Clopidogrel (lifelong)	Aspirin (lifelong) & dipyridamole (lifelong)
Peripheral arterial disease	Clopidogrel (lifelong)	Asprin (lifelong)

*the guidelines for TIA are based on the 2012 Royal College of Physicians National clinical guideline for stroke. These guidelines corrected the anomaly where patients who've had a stroke were given clopidogrel, but those who'd suffered a TIA were given aspirin + dipyridamole. Please see the link for more details (section 5.5)

External Link

[Royal College of Physicians](#)

2012 National clinical guideline for stroke

Aortic dissection

Stanford classification

- type A - ascending aorta, 2/3 of cases
- type B - descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification

- type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- type II - originates in and is confined to the ascending aorta
- type III - originates in descending aorta, rarely extends proximally but will extend distally

Associations

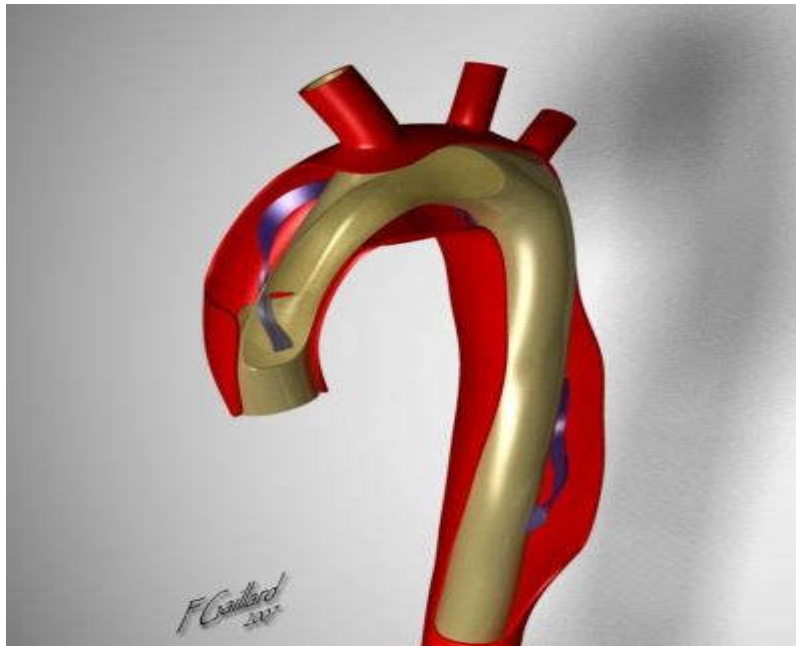
- hypertension
- trauma
- bicuspid aortic valve
- collagens: Marfan's syndrome, Ehlers-Danlos syndrome
- Turner's and Noonan's syndrome
- pregnancy
- syphilis

Complications of backward tear

- aortic incompetence/regurgitation
- MI: inferior pattern often seen due to right coronary involvement

Complications of forward tear

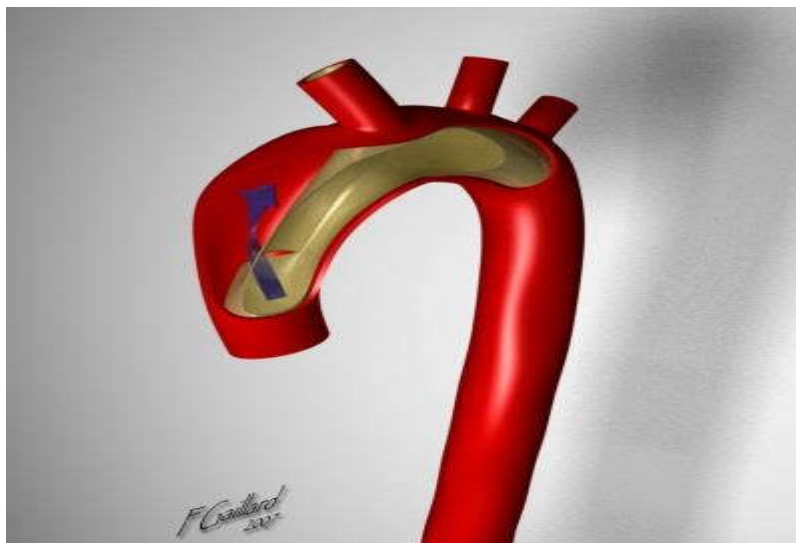
- unequal arm pulses and BP
- stroke
- renal failure



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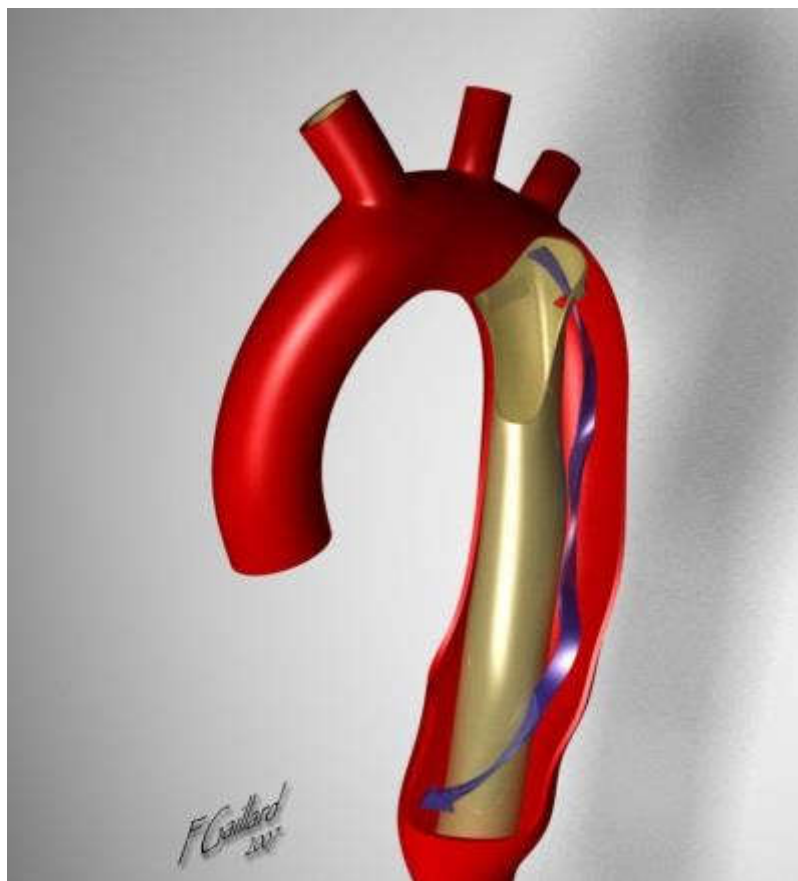
Stanford type A / DeBakey type I



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Stanford type A / DeBakey type II



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Stanford type B / DeBakey type III

Aortic dissection: management

Stanford classification

- type A - ascending aorta, 2/3 of cases
- type B - descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification

- type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- type II - originates in and is confined to the ascending aorta
- type III - originates in descending aorta, rarely extends proximally but will extend distally

Type A

- surgical management, but blood pressure should be controlled to a target systolic of 100-120 mmHg whilst awaiting intervention

Type B*

- conservative management
- bed rest
- reduce blood pressure IV labetalol to prevent progression



© Image used on license from [Radiopaedia](#)

An intraluminal tear has formed a 'flap' that can be clearly seen in the ascending aorta. This is a Stanford type A dissection



© Image used on license from [Radiopaedia](#)



Stanford type B dissection, seen in the descending aorta

*endovascular repair of type B aortic dissection may have a role in the future

External Link

[American Heart Association](#)

2010 Acute Aortic Dissection, Clinician Update.

Aortic regurgitation

Features

- early diastolic murmur
- collapsing pulse
- wide pulse pressure
- mid-diastolic Austin-Flint murmur in severe AR - due to partial closure of the anterior mitral valve cusps caused by the regurgitation streams

Causes (due to valve disease)

- rheumatic fever
- infective endocarditis
- connective tissue diseases e.g. RA/SLE
- bicuspid aortic valve

Causes (due to aortic root disease)

- aortic dissection.
- spondylarthropathies (e.g. ankylosing spondylitis).
- hypertension.
- syphilis.
- Marfan's, Ehler-Danlos syndrome.

Aortic stenosis

Features of severe aortic stenosis

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in older patients > 65 years)
- bicuspid aortic valve (most common cause in younger patients < 65 years)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 40 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement.

External Link

[European Society of Cardiology](#)

2012 Valvular heart disease guidelines

[American Heart Association](#)

Guidelines on the management of valvular heart disease

Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy.

Pathophysiology

- inherited in an autosomal dominant pattern with variable expression
- the right ventricular myocardium is replaced by fatty and fibrofatty tissue
- around 50% of patients have a mutation of one of the several genes which encode components of desmosome

Presentation

- palpitations
- syncope
- sudden cardiac death

Investigation

- ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- magnetic resonance imaging is useful to show fibrofatty tissue

Management

- drugs: sotalol is the most widely used antiarrhythmic
- catheter ablation to prevent ventricular tachycardia
- implantable cardioverter-defibrillator

Naxos disease

- an autosomal recessive variant of ARVC
- a triad of ARVC, palmoplantar keratosis, and woolly hair

Eternal Link

[Life in the fast lane](#)

Arrhythmogenic right ventricular cardiomyopathy EC

Atrial fibrillation: a very basic introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. It is very common, being present in around 5% of patients over aged 70-75 years and 10% of patients aged 80-85 years. Whilst uncontrolled atrial fibrillation can result in symptomatic palpitations and inefficient cardiac function probably the most important aspect of managing patients with AF is reducing the increased risk of stroke which is present in these patients.

Types of atrial fibrillation

AF may be classified as either first detected episode, paroxysmal, persistent or permanent.

- first detected episode (irrespective of whether it is symptomatic or self-terminating)
- recurrent episodes, when a patient has 2 or more episodes of AF. If episodes of AF terminate spontaneously then the term **paroxysmal AF** is used. Such episodes last less than 7 days (typically < 24 hours). If the arrhythmia is not self-terminating then the term **persistent AF** is used. Such episodes usually last greater than 7 days
- in **permanent AF** there is continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate. Treatment goals are therefore rate control and anticoagulation if appropriate

Symptoms and signs

Symptoms:

- palpitations
- dyspnoea
- chest pain

Signs

- an irregularly irregular pulse

Investigations

An ECG is essential to make the diagnosis as other conditions can give an irregular pulse, such as ventricular ectopics or sinus arrhythmia.

Management

There are two key parts of managing patients with AF:

1. Rate/rhythm control
2. Reducing stroke risk

Rate vs. rhythm control

There are two main strategies employed in dealing with the arrhythmia element of atrial fibrillation:

- **rate control:** accept that the pulse will be irregular, but slow the rate down to avoid negative effects on cardiac function
- **rhythm control:** try to get the patient back into, and maintain, normal sinus rhythm. This is termed cardioversion. Drugs (pharmacological cardioversion) and synchronised DC electrical shocks (electrical cardioversion) may be used for this purpose

For many years the predominant approach was to try and maintain a patient in sinus rhythm. This approach changed in the early 2000's and now the majority of patients are managed with a rate control strategy. NICE advocate using a rate control strategy except in a number of specific situations such as coexistent heart failure, first onset AF or where there is an obvious reversible cause.

Rate control

◆A **beta-blocker** or a **rate-limiting calcium channel blocker** (e.g. diltiazem) is used first-line to control the rate in AF.

◆If one drug does not control the rate adequately NICE recommend combination therapy with any 2 of the following:

- betablocker
- diltiazem
- digoxin

Rhythm control

◆As mentioned above there are a subgroup of patients for whom a rhythm control strategy should be tried first. Other patients may have had a rate control strategy initially but switch to rhythm control if symptoms/heart rate fails to settle.

◆When considering cardioversion it is very important to remember that the moment a patient switches from AF to sinus rhythm presents the highest risk for embolism leading to stroke. Imagine the thrombus formed in the fibrillating atrium suddenly being pushed out when sinus rhythm is restored. For this reason patients must either of had a short duration of symptoms (less than 48 hours) or be anticoagulated for a period of time prior to attempting cardioversion.

Reducing stroke risk

Some patients with AF are at a very low risk of stroke whilst others are at a very significant risk. Clinicians use risk stratifying tools such as the **CHA₂DS₂-VASc** score to determine the most appropriate anticoagulation strategy.

	Risk factor	Points
C	Congestive heart failure	1
H	Hypertension (or treated hypertension)	1
A₂	Age $\geq$ 75 years	2
	Age 65-74 years	1
D	Diabetes	1
S₂	Prior Stroke or TIA	2
V	Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
S	Sex (female)	1

The table below shows a suggested anticoagulation strategy based on the score:

Score	Anticoagulation
0	No treatment
1	Males: Consider anticoagulation Females: No treatment (this is because their score of 1 is only reached due to their gender)
2 or more	Offer anticoagulation

NICE recommend that we offer patients a choice of anticoagulation, including warfarin and the novel oral anticoagulants (NOACs).

Atrial fibrillation: anticoagulation

NICE updated their guidelines on the management of atrial fibrillation (AF) in 2014. They suggest using the **CHA₂DS₂-VASc** score to determine the most appropriate anticoagulation strategy. This scoring system superseded the CHADS₂ score.

	Risk factor	Points
C	Congestive heart failure	1
H	Hypertension (or treated hypertension)	1
A₂	Age $\geq$ 75 years	2
	Age 65-74 years	1
D	Diabetes	1
S₂	Prior Stroke or TIA	2
V	Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
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The table below shows a suggested anticoagulation strategy based on the score:

Score	Anticoagulation
0	No treatment
1	Males: Consider anticoagulation Females: No treatment (this is because their score of 1 is only reached due to their gender)
2 or more	Offer anticoagulation

◆NICE recommend that we offer patients a choice of anticoagulation, including warfarin and the novel oral anticoagulants (NOACs). There are complicated rules surrounding which NOAC is licensed for which risk factor - these can be found in the NICE guidelines. Aspirin is no longer recommended for reducing stroke risk in patients with AF

◆Doctors have always thought carefully about the risk/benefit profile of starting someone on warfarin. A history of falls, old age, alcohol excess and a history of previous bleeding are common things that make us consider whether warfarinisation is in the best interests of the patient. NICE now recommend we formalise this risk assessment using the HASBLED scoring system.

Chapter: cardiology

	Risk factor	Points
H	Hypertension, uncontrolled, systolic BP > 160 mmHg	1
A	Abnormal renal function (dialysis or creatinine > 200) Or Abnormal liver function (cirrhosis, bilirubin > 2 times normal, ALT/AST/ALP > 3 times normal)	1 for any renal abnormalities 1 for any liver abnormalities
S	Stroke, history of	1
B	Bleeding, history of bleeding or tendency to bleed	1
L	Labile INRs (unstable/high INRs, time in therapeutic range < 60%)	1
E	Elderly (> 65 years)	1
D	Drugs Predisposing to Bleeding (Antiplatelet agents, NSAIDs) Or Alcohol Use (>8 drinks/week)	1 for drugs 1 for alcohol

There are no formal rules on how we act on the HAS-BLED score although a score of ≥ 3 indicates a 'high risk' of bleeding, defined as intracranial haemorrhage, hospitalisation, haemoglobin decrease >2 g/L, and/or transfusion.

External Link

[European Society of Cardiology](#)

2012 Atrial fibrillation focused update

[NICE/RCP](#)

2014 Atrial fibrillation guidelines

[NICE](#)

2008 Stroke guidelines

Atrial fibrillation: cardioversion

◆ There are two scenarios where cardioversion may be used in atrial fibrillation:

- electrical cardioversion as an emergency if the patient is haemodynamically unstable
- electrical or pharmacological cardioversion as an elective procedure where a rhythm control strategy is preferred.

◆ The notes below refer to cardioversion being used in the elective scenario for rhythm control. The wording of the 2014 NICE guidelines is as follows:

offer rate or rhythm control if the onset of the arrhythmia is less than 48 hours, and start rate control if it is more than 48 hours or is uncertain

Onset < 48 hours

If the atrial fibrillation (AF) is definitely of less than 48 hours onset patients should be heparinised. Patients who have risk factors for ischaemic stroke should be put on lifelong oral anticoagulation. Otherwise, patients may be cardioverted using either:

- electrical - 'DC cardioversion'
- pharmacology - amiodarone if structural heart disease, flecainide or amiodarone in those without structural heart disease

Following electrical cardioversion if AF is confirmed as being less than 48 hours duration then further anticoagulation is unnecessary

Onset > 48 hours

◆ If the patient has been in AF for more than 48 hours then anticoagulation should be given for at least 3 weeks prior to cardioversion. An alternative strategy is to perform a transoesophageal echo (TOE) to exclude a left atrial appendage (LAA) thrombus. If excluded patients may be heparinised and cardioverted immediately.

◆ NICE recommend electrical cardioversion in this scenario, rather than pharmacological.

◆ If there is a high risk of cardioversion failure (e.g. Previous failure or AF recurrence) then it is recommend to have at least 4 weeks amiodarone or sotalol prior to electrical cardioversion

◆ Following electrical cardioversion patients should be anticoagulated for at least 4 weeks. After this time decisions about anticoagulation should be taken on an individual basis depending on the risk of recurrence.

External Link

[NICE](#)

2014 Atrial fibrillation guidelines

[European Society of Cardiology](#)

2012 Atrial fibrillation guidelines

Atrial fibrillation: classification

An attempt was made in the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines to simplify and clarify the classification of atrial fibrillation (AF).

It is recommended that AF be classified into 3 patterns:

- first detected episode (irrespective of whether it is symptomatic or self-terminating)
- recurrent episodes, when a patient has 2 or more episodes of AF. If episodes of AF terminate spontaneously then the term **paroxysmal AF** is used. Such episodes last less than 7 days (typically < 24 hours). If the arrhythmia is not self-terminating then the term **persistent AF** is used. Such episodes usually last greater than 7 days
- in **permanent AF** there is continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate. Treatment goals are therefore rate control and anticoagulation if appropriate

External Link

[NICE](#)

2014 Atrial fibrillation guidelines

Atrial fibrillation: pharmacological cardioversion

♦The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines

♦Agents with proven efficacy in the pharmacological cardioversion of atrial fibrillation:

- amiodarone
- flecainide (if no structural heart disease)
- others (less commonly used in UK): quinidine, dofetilide, ibutilide, propafenone

♦Less effective agents:

- beta-blockers (including sotalol)
- calcium channel blockers
- digoxin
- disopyramide
- procainamide

External Link

[NICE](#)

2014 NICE guidelines

[European Society of Cardiology](#)

2012 Atrial fibrillation guidelines

Atrial fibrillation: post-stroke

◆NICE issued guidelines on atrial fibrillation (AF) in 2006. They included advice on the management of patients with AF who develop a stroke or transient-ischaemic attack (TIA).

◆**Recommendations include:**

- following a stroke or TIA warfarin should be given as the anticoagulant of choice. Aspirin/dipyridamole should only be given if needed for the treatment of other comorbidities
- in acute stroke patients, in the absence of haemorrhage, anticoagulation therapy should be commenced after 2 weeks. If imaging shows a very large cerebral infarction then the initiation of anticoagulation should be delayed

External Link:

[NICE](#)

2006 Atrial fibrillation guidelines

Atrial fibrillation: rate control and maintenance of sinus rhythm

● The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines

◆ Agents used to control rate in patients with atrial fibrillation

- beta-blockers
- calcium channel blockers
- digoxin (not considered first-line anymore as they are less effective at controlling the heart rate during exercise. However, they are the preferred choice if the patient has coexistent heart failure)

◆ Agents used to maintain sinus rhythm in patients with a history of atrial fibrillation

- sotalol
- amiodarone
- flecainide
- others (less commonly used in UK): disopyramide, dofetilide, procainamide, propafenone, quinidine

The table below indicates some of the factors which may be considered when considering either a rate control or rhythm control strategy

Factors favouring rate control	Factors favouring rhythm control
Older than 65 years History of ischaemic heart disease	Younger than 65 years Symptomatic First presentation Lone AF or AF secondary to a corrected precipitant (e.g. Alcohol) Congestive heart failure

External Link

[NICE/RCP](#)

2014 Atrial fibrillation guidelines

[European Society of Cardiology](#)

2016 Atrial fibrillation guidelines

Atrial flutter

Atrial flutter is a form of supraventricular tachycardia characterised by a succession of rapid atrial depolarisation waves.

ECG findings:

- 'sawtooth' appearance
- as the underlying atrial rate is often around 300/min the ventricular or heart rate is dependent on the degree of AV block. For example if there is 2:1 block the ventricular rate will be 150/min
- flutter waves may be visible following carotid sinus massage or adenosine

Management

- is similar to that of atrial fibrillation although medication may be less effective
- atrial flutter is more sensitive to cardioversion however so lower energy levels may be used
- radiofrequency ablation of the tricuspid valve isthmus is curative for most patients

External Link

[Life in the Fast Lane](#)

Atrial Flutter

Atrial myxoma

Overview

- 75% occur in left atrium
- more common in females.

Features

- systemic: dyspnoea, fatigue, weight loss, fever, clubbing
- emboli
- atrial fibrillation
- mid-diastolic murmur, 'tumour plop'
- echo: pedunculated heterogeneous mass typically attached to the fossa ovalis region of the interatrial septum.

Beta-blockers

Beta-blockers are an important class of drug used mainly in the management of cardiovascular disorders.

Indications:

- angina
- post-myocardial infarction
- heart failure: beta-blockers were previously avoided in heart failure but there is now strong evidence that certain beta-blockers improve both symptoms and mortality
- arrhythmias: beta-blockers have now replaced digoxin as the rate-control drug of choice in atrial fibrillation
- hypertension: the role of beta-blockers has diminished in recent years due to a lack of evidence in terms of reducing stroke and myocardial infarction.
- thyrotoxicosis
- migraine prophylaxis
- anxiety

Examples

- atenolol
- propranolol: one of the first beta-blockers to be developed. Lipid soluble therefore crosses the blood-brain barrier

Side-effects

- bronchospasm
- cold peripheries
- fatigue
- sleep disturbances, including nightmares

Contraindications

- uncontrolled heart failure
- asthma
- sick sinus syndrome
- concurrent verapamil use: may precipitate severe bradycardia

External Links

[BNF](#)

Beta-blockers

Bicuspid aortic valve

Overview

- occurs in 1-2% of the population
- usually asymptomatic in childhood
- the majority eventually develop aortic stenosis or regurgitation
- associated with a left dominant coronary circulation (the posterior descending artery arises from the circumflex instead of the right coronary artery) and Turner's syndrome
- around 5% of patients also have coarctation of the aorta

Complications

- aortic stenosis/regurgitation as above
- higher risk for aortic dissection and aneurysm formation of the ascending aorta

Bivalirudin

Bivalirudin is a reversible direct thrombin inhibitor used as an anticoagulant in the management of acute coronary syndrome

Broad complex tachycardia

Features suggesting VT rather than SVT with aberrant conduction

- AV dissociation
- fusion or capture beats
- positive QRS concordance in chest leads
- marked left axis deviation
- history of IHD
- lack of response to adenosine or carotid sinus massage
- QRS > 160 ms

External Links

[Life in the fast lane](#)

VT vs SVT

Brugada syndrome

Brugada syndrome is a form of inherited cardiovascular disease with may present with sudden cardiac death. It is inherited in an autosomal dominant fashion and has an estimated prevalence of 1:5,000-10,000. Brugada syndrome is more common in Asians.

Pathophysiology

- a large number of variants exist
- around 20-40% of cases are caused by a mutation in the SCN5A gene which encodes the myocardial sodium ion channel protein

ECG changes

- convex ST segment elevation $> 2\text{mm}$ in > 1 of V1-V3 followed by a negative T wave
- partial right bundle branch block
- changes may be more apparent following flecainide



© Image used on license from [Dr Smith, University of Minnesota](#)

ECG showing Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave

Management

- implantable cardioverter-defibrillator.

External links

[Life in the fast lane](#)

Brugada syndrome

Buerger's disease

Buerger's disease (also known as thromboangiitis obliterans) is a small and medium vessel vasculitis that is strongly associated with smoking.

Features

- extremity ischaemia: intermittent claudication, ischaemic ulcers etc
- superficial thrombophlebitis
- Raynaud's phenomenon

Cardiac catheterisation and oxygen saturation levels

Questions regarding cardiac catheterisation and oxygen saturation levels can seem daunting at first but a few simple rules combined with logical deduction can usually produce the answer.

Let's start with the basics:

- deoxygenated blood returns to the right side of the heart via the superior vena cava (SVC) and inferior vena cava (IVC). It has an oxygen saturation level of around **70%**. The right atrium (RA), right ventricle (RV) and pulmonary artery (PA) normally have oxygen saturation levels of around 70%
- the lungs oxygenate the blood to a level of around **98-100%**. The left atrium (LA), left ventricle (LV) and aorta should all therefore have oxygen saturation levels of 98-100%

♦The table below shows the **oxygen saturations** that would be expected in different scenarios:

Diagnosis & notes	RA	RV	PA	LA	LV	Aorta
Normal	70%	70%	70%	100%	100%	100%
Atrial septal defect (ASD) The oxygenated blood in the LA mixes with the deoxygenated blood in the RA, resulting in intermediate levels of oxygenation from the RA onwards	85%	85%	85%	100%	100%	100%

Chapter: cardiology

Ventricular septal defect (VSD) The oxygenated blood in the LV mixes with the deoxygenated blood in the RV, resulting in intermediate levels of oxygenation from the RV onwards. The RA blood remains deoxygenated	70%	85%	85%	100%	100%	100%
Patent ductus arteriosus (PDA) Remember, a PDA connects the higher pressure aorta with the lower pressure PA. This results in only the PDA having intermediate oxygenation levels	70%	70%	85%	100%	100%	100%
VSD with Eisenmenger's	70%	70%	70%	100%	85%	85%
PDA with Eisenmenger's	70%	70%	70%	100%	100%	85%
ASD with Eisenmenger's	70%	70%	70%	85%	85%	85%

Cardiac enzymes and protein markers

Interpretation of the various cardiac enzymes has now largely been superseded by the introduction of troponin T and I. Questions still however commonly appear in exams.

Key points for the exam

- myoglobin is the first to rise
- CK-MB is useful to look for reinfarction as it returns to normal after 2-3 days (troponin T remains elevated for up to 10 days)

	Begins to rise	Peak value	Returns to normal
Myoglobin	1-2 hours	6-8 hours	1-2 days
CK-MB	2-6 hours	16-20 hours	2-3 days
CK	4-8 hours	16-24 hours	3-4 days
Trop T	4-6 hours	12-24 hours	7-10 days
AST	12-24 hours	36-48 hours	3-4 days
LDH	24-48 hours	72 hours	8-10 days

External Links

[UPMC Dept. Pathology](#)

Diagram of cardiac enzymes

[Patient.info](#)

Cardiac enzymes and protein markers review

Cardiac tamponade

Features

- dyspnoea
- raised JVP, with an absent Y descent - this is due to the limited right ventricular filling
- tachycardia
- hypotension
- muffled heart sounds
- pulsus paradoxus
- Kussmaul's sign (much debate about this)
- ECG: electrical alternans

The key differences between constrictive pericarditis and cardiac tamponade are summarized in the table below:

	Cardiac tamponade	Constrictive pericarditis
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign	Rare	Present
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

Cardiomyopathies: key points

The old classification of dilated, restricted and hypertrophic cardiomyopathy has been largely abandoned due to the high degree of overlap. The latest classification of cardiomyopathy by the WHO and American Heart Association reflect this.

The tables below shows a very limited set of exam related facts for the various cardiomyopathies:

Primary cardiomyopathies - predominately involving the heart

Genetic - both conditions listed below are autosomal dominant. An implantable cardioverter-defibrillator is often inserted to reduce the incidence of sudden cardiac death.

Type of cardiomyopathy	Selected points
Hypertrophic obstructive cardiomyopathy	Leading cause of sudden cardiac death in young athletes Usually due to a mutation in the gene encoding β -myosin heavy chain protein Common cause of sudden death Echo findings include MR, systolic anterior motion (SAM) of the anterior mitral valve and asymmetric septal hypertrophy
Arrhythmogenic right ventricular dysplasia	Right ventricular myocardium is replaced by fatty and fibrofatty tissue Around 50% of patients have a mutation of one of the several genes which encode components of desmosome ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex

Mixed - rather confusingly most of the causes of dilated and restrictive cardiomyopathy are now listed separately in the 'secondary' causes. This category serves as a reminder that many patients will have a genetic predisposition to cardiomyopathy which is then triggered by the secondary process, hence the 'mixed' category.

Chapter: cardiology

Type of cardiomyopathy	Selected causes/points
Dilated cardiomyopathy	Classic causes include: • alcohol • Cocksackie B virus • wet beri beri • doxorubicin
Restrictive cardiomyopathy	Classic causes include: • amyloidosis • post-radiotherapy • Loeffler's endocarditis

Acquired

Type of cardiomyopathy	Selected points
Peripartum cardiomyopathy	Typical develops between last month of pregnancy and 5 months post-partum More common in older women, greater parity and multiple gestations
Takotsubo cardiomyopathy	'Stress'-induced cardiomyopathy e.g. patient just found out family member dies then develops chest pain and features of heart failure Transient, apical ballooning of the myocardium Treatment is supportive

Secondary cardiomyopathies- pathological myocardial involvement as part of a generalized systemic disorder

Type of cardiomyopathy	Selected causes/points
Infective	Cocksackie B virus Chagas disease
Infiltrative	Amyloidosis
Storage	Haemochromatosis
Toxicity	Doxorubicin Alcoholic cardiomyopathy
Inflammatory (granulomatous)	Sarcoidosis
Endocrine	Diabetes mellitus Thyrotoxicosis Acromegaly
Neuromuscular	Friedreich's ataxia Duchenne-Becker muscular dystrophy Myotonic dystrophy
Nutritional deficiencies	Beriberi (thiamine)
Autoimmune	Systemic lupus erythematosus

Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a form of inherited cardiac disease associated with sudden cardiac death. It is inherited in an autosomal dominant fashion and has a prevalence of around 1:10,000.

Pathophysiology

- the most common cause is a defect in the ryanodine receptor (RYR2) which is found in the myocardial sarcoplasmic reticulum

Features

- exercise or emotion induced polymorphic ventricular tachycardia resulting in syncope
- sudden cardiac death
- symptoms generally develop before the age of 20 years

Management

- beta-blockers
- implantable cardioverter-defibrillator

Centrally acting antihypertensive

Examples of centrally acting antihypertensives include:

- methyldopa: used in the management of hypertension during pregnancy
- moxonidine: used in the management of essential hypertension when conventional antihypertensives have failed to control blood pressure
- clonidine: the antihypertensive effect is mediated through stimulating alpha-2 adrenoceptors in the vasomotor centre

Chest pain: assessment of patients with suspected cardiac chest pain

NICE issued guidelines in 2010 on the 'Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin'.

Below is a brief summary of the key points. Please see the link for more details.

Patients presenting with acute chest pain

Immediate management of suspected acute coronary syndrome (ACS):

- glyceryl trinitrate
- aspirin 300mg. NICE do not recommend giving other antiplatelet agents (i.e. Clopidogrel) outside of hospital
- do not routinely give oxygen, only give if sats < 94%*
- perform an ECG as soon as possible but do not delay transfer to hospital. A normal ECG does not exclude ACS

Referral

- current chest pain or chest pain in the last 12 hours with an abnormal ECG: emergency admission
- chest pain 12-72 hours ago: refer to hospital the same-day for assessment
- chest pain > 72 hours ago: perform full assessment with ECG and troponin measurement before deciding upon further action.

***NICE suggest the following in terms of oxygen therapy:**

- do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:
- people with oxygen saturation (SpO₂) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO₂ of 94-98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO₂ of 88-92% until blood gas analysis is available.

Patients presenting with stable chest pain

With all due respect to NICE the guidelines for assessment of patients with stable chest pain are rather complicated. They suggest an approach where the risk of a patient having coronary artery disease (CAD) is calculated based on their symptoms (whether they have typical angina, atypical angina or non-anginal chest pain), age, gender and risk factors.

NICE define anginal pain as the following:

1. constricting discomfort in the front of the chest, neck, shoulders, jaw or arms
2. precipitated by physical exertion
3. relieved by rest or GTN in about 5 minutes.

Chapter: cardiology

♦ patients with all 3 features have typical angina.

♦ patients with 2 of the above features have **atypical** angina.

♦ patients with 1 or none of the above features have non-anginal chest pain.

The risk tables are not reproduced here but can be found by clicking on the link.

♦If patients have typical anginal symptoms and a risk of CAD is greater than 90% then no further diagnostic testing is required. It should be noted that all men over the age of 70 years who have typical anginal symptoms fall into this category.

♦For patients with an estimated risk of 10-90% the following investigations are recommended.
Note the absence of the exercise tolerance test:

Estimated likelihood of CAD	Diagnostic testing
61-90%	Coronary angiography
30-60%	Functional imaging, for example: • myocardial perfusion scan with SPECT • stress echocardiography • first-pass contrast-enhanced magnetic resonance (MR) perfusion • MR imaging for stress-induced wall motion abnormalities.
10-29%	CT calcium scoring

External links

[NICE](#)

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

Cholestyramine

♦Cholestyramine is a bile acid sequestrant used in the management of hyperlipidaemia. It decreases bile acid reabsorption in the small intestine, therefore upregulating the amount of cholesterol that is converted to bile acid. The main effect it has on the lipid profile is to reduce LDL cholesterol. It is also occasionally used in Crohn's disease for treatment resistant diarrhoea.

Adverse effects

- abdominal cramps and constipation
- decreases absorption of fat-soluble vitamins
- cholesterol gallstones
- may raise level of triglycerides

Chronic kidney disease: anaemia

Patients with chronic kidney disease (CKD) may develop anaemia due to a variety of factors, the most significant of which is reduced erythropoietin levels. This is usually a normochromic normocytic anaemia and becomes apparent when the GFR is less than 35 ml/min (other causes of anaemia should be considered if the GFR is > 60 ml/min). Anaemia in CKD predisposes to the development of left ventricular hypertrophy - associated with a three fold increase in mortality in renal patients

Causes of anaemia in renal failure:

- reduced erythropoietin levels - the most significant factor
- reduced erythropoiesis due to toxic effects of uraemia on bone marrow
- reduced absorption of iron
- anorexia/nausea due to uraemia
- reduced red cell survival (especially in haemodialysis)
- blood loss due to capillary fragility and poor platelet function
- stress ulceration leading to chronic blood loss.

Management

- the 2011 NICE guidelines suggest a target haemoglobin of 10 - 12 g/dl
- determination and optimisation of iron status should be carried out prior to the administration of erythropoiesis-stimulating agents (ESA). Many patients, especially those on haemodialysis, will require IV iron
- ESAs such as erythropoietin and darbepoetin should be used in those 'who are likely to benefit in terms of quality of life and physical function'

External links

[NICE](#)

2015 Chronic kidney disease: managing anaemia

[The Renal Association](#)

CKD anaemia guidelines

Coarctation of the aorta

Coarctation of the aorta describes a congenital narrowing of the descending aorta.

Overview

- more common in males (despite association with Turner's syndrome)

Features

- infancy: heart failure
- adult: hypertension
- radio-femoral delay
- mid systolic murmur, maximal over back
- apical click from the aortic valve
- notching of the inferior border of the ribs (due to collateral vessels) is not seen in young children.

Associations

- Turner's syndrome
 - bicuspid aortic valve
 - berry aneurysms
 - neurofibromatosis
-

Complete heart block

Features:

- syncope
- heart failure
- regular bradycardia (30-50 bpm)
- wide pulse pressure
- JVP: cannon waves in neck
- variable intensity of S1

Types of heart block

First degree heart block:

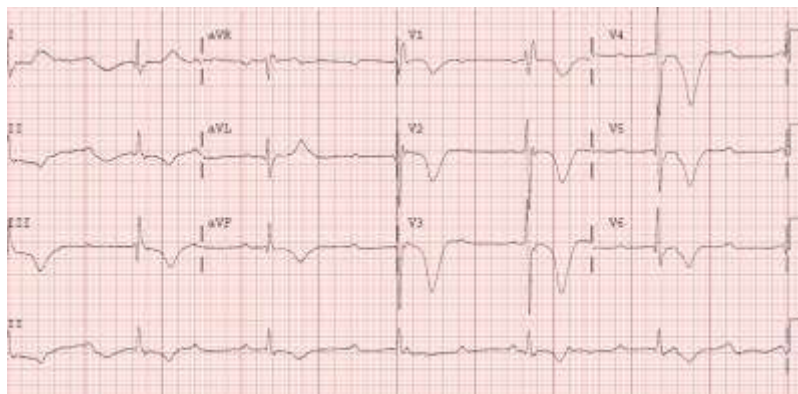
- PR interval > 0.2 seconds

Second degree heart block:

- type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs
- type 2 (Mobitz II): PR interval is constant but the P wave is often not followed by a QRS complex.

Third degree (complete) heart block:

- there is no association between the P waves and QRS complexes



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ECG showing third degree (complete) heart block

External Links

[ECGPedia](#)

AV conduction problems

Congenital heart disease: types

Acyanotic - most common causes

- ventricular septal defects (VSD) - most common, accounts for 30%
- atrial septal defect (ASD)
- patent ductus arteriosus (PDA)
- coarctation of the aorta
- aortic valve stenosis.

♦ *VSDs are more common than ASDs. However, in adult patients ASDs are the more common new diagnosis as they generally presents later.*

Chapter: cardiology

Cyanotic - most common causes:

- tetralogy of Fallot
- transposition of the great arteries (TGA)
- tricuspid atresia

♦Fallot's is more common than TGA. However, at birth TGA is the more common lesion as patients with Fallot's generally presenting at around 1-2 months

♦The presence of cyanosis in pulmonary valve stenosis depends very much on the severity and any other coexistent defects.

Constrictive pericarditis

Causes

- any cause of pericarditis
- particularly TB

Features

- dyspnoea
- right heart failure: elevated JVP, ascites, oedema, hepatomegaly
- JVP shows prominent x and y descent
- pericardial knock - loud S3
- Kussmaul's sign is positive

CXR

- pericardial calcification.

The key differences between constrictive pericarditis and cardiac tamponade are summarized in the table below:

	Cardiac tamponade	Constrictive pericarditis
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign*	Rare	Present
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

*a paradoxical rise in JVP during inspiration

Coronary circulation

Arterial supply of the heart

- left aortic sinus → left coronary artery (LCA)
- right aortic sinus → right coronary artery (RCA)
- LCA → LAD + circumflex
- RCA → posterior descending
- RCA supplies SA node in 60%, AV node in 90%

Venous drainage of the heart

- coronary sinus drains into the right atrium
-

Diabetes mellitus: hypertension management

NICE recommend the following blood pressure targets for diabetics:

- if end-organ damage (e.g. renal disease, retinopathy) < 130/80 mmHg
- otherwise < 140/80 mmHg

A 2013 Cochrane review casted doubt on the wisdom of lower blood pressure targets for patients with diabetes. It compared patients who had tight blood pressure control (targets < 130/85 mmHg) with more relaxed control (< 140-160/90-100 mmHg). Patients who were more tightly controlled had a slightly reduced rate of stroke but otherwise outcomes were not significantly different.

Because ACE-inhibitors have a renoprotective effect in diabetes they are the first-line antihypertensives recommended for NICE. Patients of African or Caribbean family origin should be offered an ACE-inhibitor plus either a thiazide diuretic or calcium channel blocker. Further management then reverts to that of non-diabetic patients, as discussed earlier in the module.

Remember that autonomic neuropathy may result in more postural symptoms in patients taking antihypertensive therapy.

The routine use of beta-blockers in uncomplicated hypertension should be avoided, particularly when given in combination with thiazides, as they may cause insulin resistance, impair insulin secretion and alter the autonomic response to hypoglycaemia.

Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) basics

- dilated heart leading to systolic (+/- diastolic) dysfunction
- all 4 chambers affected but LV more so than RV
- features include arrhythmias, emboli, mitral regurgitation
- absence of congenital, valvular or ischaemic heart disease.

Causes often considered separate entities:

- alcohol: may improve with thiamine
- postpartum
- hypertension

Other causes

- inherited (see below)
- infections e.g. Coxsackie B, HIV, diphtheria, parasitic
- endocrine e.g. Hyperthyroidism
- infiltrative* e.g. Haemochromatosis, sarcoidosis
- neuromuscular e.g. Duchenne muscular dystrophy
- nutritional e.g. Kwashiorkor, pellagra, thiamine/selenium deficiency
- drugs e.g. Doxorubicin

Inherited dilated cardiomyopathy

- around a third of patients with DCM are thought to have a genetic predisposition
- a large number of heterogeneous defects have been identified
- the majority of defects are inherited in an autosomal dominant fashion although other patterns of inheritance are seen

*these causes may also lead to restrictive cardiomyopathy

Down syndrome: features

Clinical features

- face: upslanting palpebral fissures, epicanthic folds, Brushfield spots in iris, protruding tongue, small ears, round/flat face
- flat occiput
- single palmar crease, pronounced 'sandal gap' between big and first toe
- hypotonia
- congenital heart defects (40-50%, see below)
- duodenal atresia
- Hirschsprung's disease

Cardiac complications

- multiple cardiac problems may be present
- endocardial cushion defect (c. 40%, also known as atrioventricular septal canal defects)
- ventricular septal defect (c. 30%)
- secundum atrial septal defect (c. 10%)
- tetralogy of Fallot (c. 5%)
- isolated patent ductus arteriosus (c. 5%)

Later complications

- subfertility: males are almost always infertile due to impaired spermatogenesis. Females are usually subfertile, and have an increased incidence of problems with pregnancy and labour
- learning difficulties
- short stature
- repeated respiratory infections (+hearing impairment from glue ear)
- acute lymphoblastic leukaemia
- hypothyroidism
- Alzheimer's
- atlantoaxial instability

DVLA: cardiovascular disorders

The guidelines below relate to car/motorcycle use unless specifically stated. For obvious reasons, the rules relating to drivers of heavy goods vehicles tend to be much stricter

Specific rules:

- hypertension - can drive unless treatment causes unacceptable side effects, no need to notify DVLA. If Group 2 Entitlement the disqualifies from driving if resting BP consistently 180 mmHg systolic or more and/or 100 mm Hg diastolic or more
- angioplasty (elective) - 1 week off driving
- CABG - 4 weeks off driving
- acute coronary syndrome- 4 weeks off driving, 1 week if successfully treated by angioplasty
- angina - driving must cease if symptoms occur at rest/at the wheel
- pacemaker insertion - 1 week off driving
- implantable cardioverter-defibrillator (ICD): if implanted for sustained ventricular arrhythmia: cease driving for 6 months. If implanted prophylactically then cease driving for 1 month. Having an ICD results in a permanent bar for Group 2 drivers
- successful catheter ablation for an arrhythmia- 2 days off driving
- aortic aneurysm of 6cm or more - notify DVLA. Licensing will be permitted subject to annual review. An aortic diameter of 6.5 cm or more disqualifies patients from driving
- heart transplant: DVLA do not need to be notified

External Links

[DVLA](#)

Cardiovascular disorder guidelines

Ebstein's anomaly

Ebstein's anomaly is a congenital heart defect characterised by low insertion of the tricuspid valve resulting in a large atrium and small ventricle. It is sometimes referred to as 'atrialisation' of the right ventricle.

Associations

- tricuspid incompetence (pan-systolic murmur, giant V waves in JVP)
- Wolff-Parkinson White syndrome

Ebstein's anomaly may be caused by exposure to lithium in-utero

ECG: axis deviation

Causes of left axis deviation (LAD)

- left anterior hemiblock
- left bundle branch block
- Wolff-Parkinson-White syndrome* - right-sided accessory pathway
- hyperkalaemia
- congenital: ostium primum ASD, tricuspid atresia
- minor LAD in obese people

Causes of right axis deviation (RAD)

- right ventricular hypertrophy
- left posterior hemiblock
- chronic lung disease → cor pulmonale
- pulmonary embolism
- ostium secundum ASD
- Wolff-Parkinson-White syndrome* - left-sided accessory pathway
- normal in infant < 1 years old
- minor RAD in tall people

*in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation.

External Links

[Life in the Fastlane](#)

ECG Axis Interpretation

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V4-6, I, aVL	Left anterior descending or left circumflex
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Tall R waves V1-2	Usually left circumflex, also right coronary



Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

External Links

[Postgraduate Medical Journal](#)

ECG correlation with coronary anatomy

[University of Massachusetts Medical School](#)

Classic circumflex infarction ECG

ECG: digoxin

ECG features:

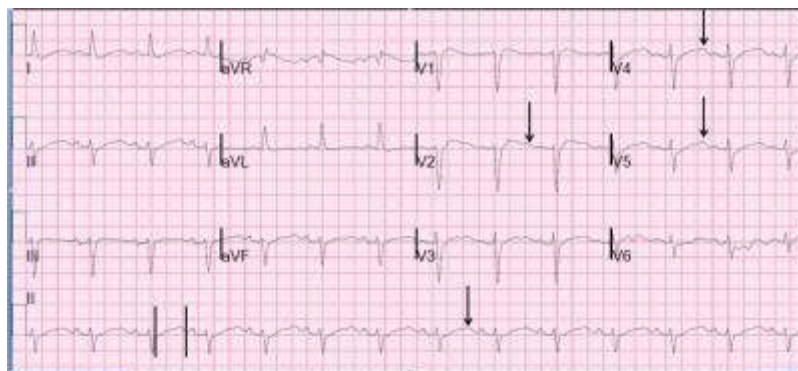
- down-sloping ST depression ('reverse tick')
- flattened/inverted T waves
- short QT interval
- arrhythmias e.g. AV block, bradycardia

ECG: hypokalaemia

ECG features of hypokalaemia

- U waves
- small or absent T waves (occasionally inversion)
- prolong PR interval
- ST depression
- long QT

The ECG below shows typical U waves. Note also the borderline PR interval.



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One registered user suggests the following rhyme

- In Hypokalaemia, U have no Pot and no T, but a long PR and a long QT

External Links

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ECG library: Hypokalaemia

ECG: hypothermia

The following ECG changes may be seen in hypothermia:

- bradycardia
- 'J' wave - small hump at the end of the QRS complex
- first degree heart block
- long QT interval
- atrial and ventricular arrhythmias

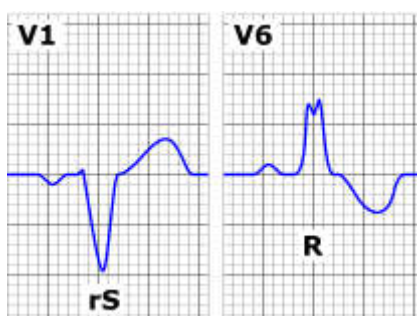
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ECG changes in hypothermia

ECG: left bundle branch block

The diagram below shows the typical features of left bundle branch block (LBBB):



One of the most common ways to remember the difference between LBBB and RBBB is WiLLiaM MaRRoW

- in LBBB there is a 'W' in V1 and a 'M' in V6
- in RBBB there is a 'M' in V1 and a 'W' in V6



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ECG showing typical features of LBBB

Causes of LBBB

- ischaemic heart disease
- hypertension
- aortic stenosis
- cardiomyopathy
- rare: idiopathic fibrosis, digoxin toxicity, hyperkalaemia

New LBBB is always pathological and may be a sign of a myocardial infarction. Diagnosing a myocardial infarction for patients with *existing* LBBB is difficult. The Sgarbossa criteria can help with this. Please see the link for more details.

External Links

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Sgarbossa criteria

ECG: normal variants

The following ECG changes are considered normal variants in an athlete:

- sinus bradycardia
- junctional rhythm
- first degree heart block
- Wenckebach phenomenon

External Links:

[Life In The Fast Lane](#)

Example of ECG changes in an athlete

ECG: P wave changes

Increased P wave amplitude

- cor pulmonale

ECG: PR interval

Causes of a prolonged PR interval

- idiopathic
- ischaemic heart disease
- digoxin toxicity
- hypokalaemia*
- rheumatic fever
- aortic root pathology e.g. abscess secondary to endocarditis
- Lyme disease
- sarcoidosis
- myotonic dystrophy

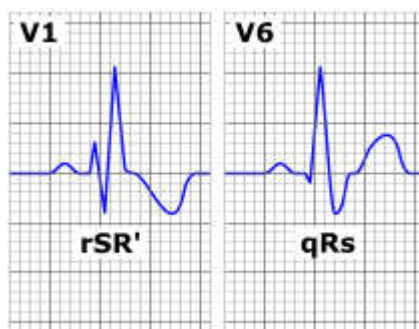
A prolonged PR interval may also be seen in athletes.

A **short PR interval** is seen in Wolff-Parkinson-White syndrome

*hyperkalaemia can rarely cause a prolonged PR interval, but this is a much less common association than hypokalaemia

ECG: right bundle branch block

Right bundle branch block is a common feature seen on ECGs.



One of the most common ways to remember the difference between LBBB and RBBB is WILLiaM MaRRoW

- in LBBB there is a 'W' in V1 and a 'M' in V6
- in RBBB there is a 'M' in V1 and a 'W' in V6.

Causes of RBBB

- normal variant - more common with increasing age
 - right ventricular hypertrophy
 - chronically increased right ventricular pressure - e.g. cor pulmonale
 - pulmonary embolism
 - myocardial infarction
 - atrial septal defect (ostium secundum)
 - cardiomyopathy or myocarditis
-

External Link

[Life In The Fast Lane](#)

Right Bundle Branch Block

ECG: ST depression

Causes of ST depression

- secondary to abnormal QRS (LVH, LBBB, RBBB)
 - ischaemia
 - digoxin
 - hypokalaemia
 - syndrome X
-

ECG: ST elevation

Causes of ST elevation:

- myocardial infarction
 - pericarditis
 - normal variant - 'high take-off'
 - left ventricular aneurysm
 - Prinzmetal's angina (coronary artery spasm)
 - rare: subarachnoid haemorrhage
-

ECG: T wave changes

Peaked T waves

- hyperkalaemia
- myocardial ischaemia

Inverted T waves

- myocardial ischaemia
- digoxin toxicity
- subarachnoid haemorrhage
- arrhythmogenic right ventricular cardiomyopathy
- pulmonary embolism ('S1Q3T3')
- Brugada syndrome

Eisenmenger's syndrome

Eisenmenger's syndrome describes the reversal of a left-to-right shunt in a congenital heart defect due to pulmonary hypertension. This occurs when an uncorrected left-to-right leads to remodeling of the pulmonary microvasculature, eventually causing obstruction to pulmonary blood and pulmonary hypertension.

Associated with

- ventricular septal defect
- atrial septal defect
- patent ductus arteriosus

Features

- original murmur may disappear
- cyanosis
- clubbing
- right ventricular failure
- haemoptysis, embolism

Management

- heart-lung transplantation is required

Exercise tolerance tests

Exercise tolerance tests (ETT, also exercise ECG) are used for a variety of indications:

- assessing patients with suspected angina - however the 2010 NICE Chest pain of recent onset guidelines do not support the use of ETTs for all patients
- risk stratifying patients following a myocardial infarction
- assessing exercise tolerance
- risk stratifying patients with hypertrophic cardiomyopathy

ETT has a sensitivity of around 80% and a specificity of 70% for ischaemic heart disease.

Heart rate:

- maximum predicted heart rate = $220 - \text{patient's age}$
- the target heart rate is at least 85% of maximum predicted to allow reasonable interpretation of

a test as low-risk or negative

Contraindications

- myocardial infarction less than 7 days ago
- unstable angina
- uncontrolled hypertension (systolic BP > 180 mmHg) or hypotension (systolic BP < 90 mmHg)
- aortic stenosis
- left bundle branch block: this would make the ECG very difficult to interpret

Stop if:

- exhaustion / patient request
- 'severe', 'limiting' chest pain
- $> 3\text{mm}$ ST depression
- $> 2\text{mm}$ ST elevation. Stop if rapid ST elevation and pain
- systolic blood pressure > 230 mmHg
- systolic blood pressure falling > 20 mmHg
- attainment of maximum predicted heart rate
- heart rate falling $> 20\%$ of starting rate
- arrhythmia develops

External Links

[British Cardiac Society](#)

Protocol for exercise tolerance test

Exercise: physiological changes

Blood pressure

- systolic increases, diastolic decreases
- leads to increased pulse pressure
- in healthy young people the increase in MABP is only slight

Cardiac output

- increase in cardiac output may be 3-5 fold
- results from venous constriction, vasodilation and increased myocardial contractility, as well as from the maintenance of right atrial pressure by an increase in venous return
- heart rate up to 3-fold increase
- stroke volume up to 1.5-fold increase

Heart failure: diagnosis

NICE issued updated guidelines on diagnosis and management in 2010. The choice of investigation is determined by whether the patient has previously had a myocardial infarction or not.

Previous myocardial infarction:

- arrange echocardiogram within 2 weeks

No previous myocardial infarction:

- measure serum natriuretic peptides (BNP)
- if levels are 'high' arrange echocardiogram within 2 weeks
- if levels are 'raised' arrange echocardiogram within 6 weeks.

Chapter: cardiology

Serum natriuretic peptides

B-type natriuretic peptide (BNP) is a hormone produced mainly by the left ventricular myocardium in response to strain. Very high levels are associated with a poor prognosis.

	BNP	NTproBNP
High levels	> 400 pg/ml (116 pmol/litre)	> 2000 pg/ml (236 pmol/litre)
Raised levels	100-400 pg/ml (29-116 pmol/litre)	400-2000 pg/ml (47-236 pmol/litre)
Normal levels	< 100 pg/ml (29 pmol/litre)	< 400 pg/ml (47 pmol/litre)

Factors which alter the BNP level:

Increase BNP levels	Decrease BNP levels
Left ventricular hypertrophy Ischaemia Tachycardia Right ventricular overload Hypoxaemia (including pulmonary embolism) GFR < 60 ml/min Sepsis COPD Diabetes Age > 70 Liver cirrhosis	Obesity Diuretics ACE inhibitors Beta-blockers Angiotensin 2 receptor blockers Aldosterone antagonists

External Links

[NICE](#)

2010 Heart failure guidelines

Heart failure: drug management

A number of drugs have been shown to improve mortality in patients with chronic heart failure:

- ACE inhibitors (SAVE, SOLVD, CONSENSUS)
- spironolactone (RALES)
- beta-blockers (CIBIS)
- hydralazine with nitrates (VHEFT-1)

◆No long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

NICE issued updated guidelines on management in 2010, key points include:

- first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**
- second-line treatment is now either an **aldosterone antagonist**, angiotensin II receptor blocker or a hydralazine in combination with a nitrate
- if symptoms persist **cardiac resynchronisation therapy** or **digoxin*** should be considered. An alternative supported by NICE in 2012 is **ivabradine**. The criteria for ivabradine include that the patient is already on suitable therapy (ACE-inhibitor, beta-blocker + aldosterone antagonist), has a heart rate > 75/min and a left ventricular fraction < 35%
- **diuretics** should be given for fluid overload
- offer **annual influenza vaccine**
- offer one-off** **pneumococcal vaccine**

♦Beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.

*digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties. Digoxin is strongly indicated if there is coexistent atrial fibrillation

**adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years

External Links

[NICE](#)

2010 Heart failure guidelines

[NICE](#)

2012 Guidance on ivabradine

Heart failure: non-drug management

Cardiac resynchronisation therapy:

- for patients with heart failure and wide QRS
- biventricular pacing
- improved symptoms and reduced hospitalisation in NYHA class III patients

Exercise training:

- improves symptoms but not hospitalisation/mortality

External Links

[NICE](#)

2010 Heart failure guidelines

[NICE](#)

Cardiac resynchronisation therapy for the treatment of heart failure

Heart failure: NYHA classification

The New York Heart Association (NYHA) classification is widely used to classify the severity of heart failure:

NYHA Class I

- no symptoms
- no limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations

NYHA Class II

- mild symptoms
- slight limitation of physical activity: comfortable at rest but ordinary activity results in fatigue, palpitations or dyspnoea

NYHA Class III

- moderate symptoms
- marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms

NYHA Class IV

- severe symptoms
- unable to carry out any physical activity without discomfort: symptoms of heart failure are present even at rest with increased discomfort with any physical activity

External Links

[NICE](#)

2010 Heart failure guidelines

Heart sounds

The first heart sound (S1) is caused by closure of the mitral and tricuspid valves whilst the second heart sound (S2) is due to aortic and pulmonary valve closure

S1

- closure of mitral and tricuspid valves
- soft if long PR or mitral regurgitation
- loud in mitral stenosis

S2

- closure of aortic and pulmonary valves
- soft in aortic stenosis
- splitting during inspiration is normal

S3 (third heart sound)

- caused by diastolic filling of the ventricle
- considered normal if < 30 years old (may persist in women up to 50 years old)
- heard in left ventricular failure (e.g. dilated cardiomyopathy), constrictive pericarditis (called a pericardial knock) and mitral regurgitation

S4 (fourth heart sound)

- may be heard in aortic stenosis, HOCM, hypertension
- caused by atrial contraction against a stiff ventricle
- in HOCM a double apical impulse may be felt as a result of a palpable S4.

Sites of auscultation

Valve	Site
Pulmonary valve	Left second intercostal space, at the upper sternal border
Aortic valve	Right second intercostal space, at the upper sternal border
Mitral valve	Left fifth intercostal space, just medial to mid clavicular line
Tricuspid valve	Left fourth intercostal space, at the lower left sternal border

The diagram below demonstrates where the various cardiac valves are best heard.

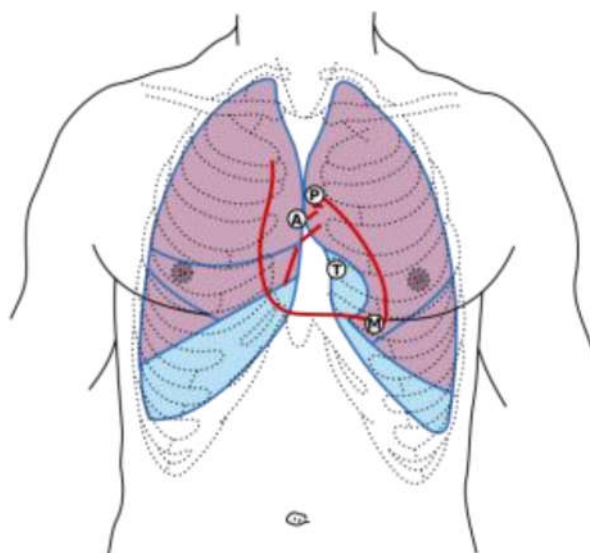


Image sourced from [Wikipedia](#)

External Links

[Wikipedia](#)

Cardiac cycle

Heart sounds: S1

S1 is caused by closure of mitral and tricuspid valves

Causes of a loud S1

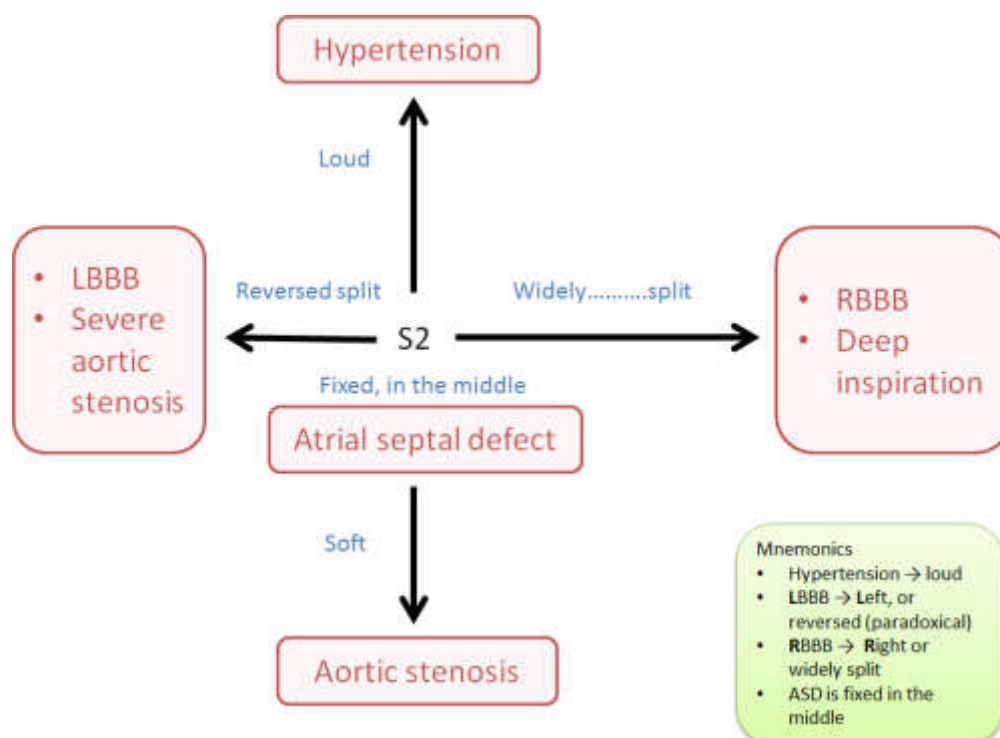
- mitral stenosis
- left to right shunts
- short PR interval, atrial premature beats
- hyperdynamic states

Causes of a quiet S1

- mitral regurgitation

Heart sounds: S2

S2 is caused by the closure of the aortic valve (A2) closely followed by that of the pulmonary valve (P2)



Chapter: cardiology

Causes of a loud S2

- hypertension: systemic (loud A2) or pulmonary (loud P2)
- hyperdynamic states
- atrial septal defect without pulmonary hypertension

Causes of a soft S2

- aortic stenosis

Causes of fixed split S2

- atrial septal defect

Causes of a widely split S2

- deep inspiration
- RBBB
- pulmonary stenosis
- severe mitral regurgitation

Causes of a reversed (paradoxical) split S2 (P2 occurs before A2)

- LBBB
- severe aortic stenosis
- right ventricular pacing
- WPW type B (causes early P2)
- patent ductus arteriosus

External Links

[YouTube](#)

S2 tutorial

HOCM: features

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. The most common defects involve a mutation in the gene encoding β -myosin heavy chain protein or myosin binding protein C. The estimated prevalence is 1 in 500.

Features

- often asymptomatic
- dyspnoea, angina, syncope
- sudden death (most commonly due to ventricular arrhythmias), arrhythmias, heart failure
- jerky pulse, large 'a' waves, double apex beat
- ejection systolic murmur: increases with Valsalva manoeuvre and decreases on squatting

Associations

- Friedreich's ataxia
- Wolff-Parkinson White

Echo - mnemonic - MR SAM ASH

- mitral regurgitation (MR)
- systolic anterior motion (SAM) of the anterior mitral valve leaflet
- asymmetric hypertrophy (ASH)

ECG

- left ventricular hypertrophy
- progressive T wave inversion
- deep Q waves
- atrial fibrillation may occasionally be seen



© Image used on license from [Dr Smith, University of Minnesota](#)

ECG showing typical changes of HOCM including LVH and T wave inversion

External Links

[RCP](#)

Investigation and treatment of hypertrophic cardiomyopathy

[American College of Cardiology Foundation \(ACCF\) and the American Heart Association \(AHA\)](#)

2011 HOCM guidelines

[European Society of Cardiology \(ESC\)](#)

2014 ESC HCOM Guidelines

HOCM: management

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. The estimated prevalence is 1 in 500.

Management

- Amiodarone
- Beta-blockers or verapamil for symptoms
- Cardioverter defibrillator
- Dual chamber pacemaker
- Endocarditis prophylaxis*

Drugs to avoid

- nitrates
- ACE-inhibitors
- inotropes

*although see the 2008 NICE guidelines on infective endocarditis prophylaxis

External Links

[RCP](#)

Investigation and treatment of hypertrophic cardiomyopathy

HOCM: prognostic factors

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. Mutations to various proteins including beta-myosin, alpha-tropomyosin and troponin T have been identified.

Septal hypertrophy causes left ventricular outflow obstruction. It is an important cause of sudden death in apparently healthy individuals.

Poor prognostic factors:

- syncope
- family history of sudden death
- young age at presentation
- non-sustained ventricular tachycardia on 24 or 48-hour Holter monitoring
- abnormal blood pressure changes on exercise

An increased septal wall thickness is also associated with a poor prognosis.

External Links

[RCP](#)

Investigation and treatment of hypertrophic cardiomyopathy

Hydralazine

Hydralazine is one of the 'older' antihypertensives and is not commonly used nowadays. It is still sometimes used for hypertension in pregnancy and for severe hypertension.

Mechanism of action:

- increases cGMP leading to smooth muscle relaxation

Contraindications:

- systemic lupus erythematosus
- ischaemic heart disease

Adverse effects:

- tachycardia
 - palpitations
 - flushing
 - fluid retention
 - headache
 - drug-induced lupus
-

Hypercalcaemia: features

Features

- 'bones, stones, groans and psychic moans', also:
- corneal calcification
- shortened QT interval on ECG
- hypertension

Hyperlipidaemia: xanthomata

Characteristic xanthomata seen in hyperlipidaemia:

Palmar xanthoma

- remnant hyperlipidaemia
- may less commonly be seen in familial hypercholesterolaemia

Eruptive xanthoma are due to high triglyceride levels and present as multiple red/yellow vesicles on the extensor surfaces (e.g. elbows, knees)

Causes of eruptive xanthoma

- familial hypertriglyceridaemia
- lipoprotein lipase deficiency

Tendon xanthoma, tuberous xanthoma, xanthelasma

- familial hypercholesterolaemia
- remnant hyperlipidaemia

Xanthelasma are also seen without lipid abnormalities

Management of xanthelasma, options include:

- surgical excision
- topical trichloroacetic acid
- laser therapy
- electrodesiccation

External Links

[DermNet NZ](#)

Xanthomas

Hypertension in pregnancy

NICE published guidance in 2010 on the management of hypertension in pregnancy. They also made recommendations on reducing the risk of hypertensive disorders developing in the first place. Women who are at high risk of developing pre-eclampsia should take aspirin 75mg od from 12 weeks until the birth of the baby. **High risk groups include:**

- hypertensive disease during previous pregnancies
- chronic kidney disease
- autoimmune disorders such as SLE or antiphospholipid syndrome
- type 1 or 2 diabetes mellitus

The classification of hypertension in pregnancy is complicated and varies. Remember, in normal pregnancy:

- blood pressure usually falls in the first trimester (particularly the diastolic), and continues to fall until 20-24 weeks
- after this time the blood pressure usually increases to pre-pregnancy levels by term

Hypertension in pregnancy is usually defined as:

- systolic > 140 mmHg or diastolic > 90 mmHg
- or an increase above booking readings of > 30 mmHg systolic or > 15 mmHg diastolic

◆After establishing that the patient is hypertensive they should be categorised into one of the following groups:

Pre-existing hypertension	Pregnancy-induced hypertension (PIH, also known as gestational hypertension)	Pre-eclampsia
●A history of hypertension before pregnancy or an elevated blood pressure > 140/90 mmHg before 20 weeks gestation ●No proteinuria, no oedema ●Occurs in 3-5% of pregnancies and is more common in older women	●Hypertension (as defined above) occurring in the second half of pregnancy (i.e. after 20 weeks) ●No proteinuria, no oedema ●Occurs in around 5-7% of pregnancies Resolves following birth (typically after one month). Women with PIH are at increased risk of future pre-eclampsia or hypertension later in life	●Pregnancy-induced hypertension in association with proteinuria (> 0.3g / 24 hours) ●Oedema may occur but is now less commonly used as a criteria ●Occurs in around 5% of pregnancies

External Links

[NICE](#)

2010 Hypertension in pregnancy

[Royal College of Physicians](#)

2013 Renal disease and hypertension in pregnancy.

Hypertension: a very basic introduction

Hypertension is one of the most common medical conditions encountered in the developed world. Whilst there is a degree of normal variation in blood pressure according to the time of day and whether we are exerting ourselves hypertension describes a chronically raised blood pressure. The main relevance of hypertension lies in the fact that it is an important risk factor for the development of cardiovascular disease such as ischaemic heart disease and stroke. Unless the blood pressure is very high it is unusual for patients to experience any symptoms.

What is a 'normal' blood pressure?

Normal blood pressure can vary widely according to age, gender and individual physiology. Most healthy people have a blood pressure between 90/60 mmHg and 140/90 mmHg.

NICE define hypertension as follows:

- a clinic reading persistently above $\geq 140/90$ mmHg, or:
- a 24 hour blood pressure average reading $\geq 135/85$ mmHg

Why do some patients have an elevated blood pressure?

Patients with hypertension may be divided into two categories. The vast majority (around 90-95%) have primary, or essential, hypertension. This is where there is no single disease causing the rise in blood pressure but rather a series of complex physiological changes which occur as we get older.

Secondary hypertension may be caused by a wide variety of endocrine, renal and other causes. The table below lists some of the conditions that may cause secondary hypertension.

Renal disease	Endocrine disorders	Other causes
• Glomerulonephritis • Chronic pyelonephritis • Adult polycystic kidney disease • Renal artery stenosis	• Primary hyperaldosteronism • Pheochromocytoma • Cushing's syndrome • Liddle's syndrome • Congenital adrenal hyperplasia (11-beta hydroxylase deficiency) • Acromegaly	• Glucocorticoids • NSAIDs • Pregnancy • Coarctation of the aorta • Combined oral contraceptive pill

Chapter: cardiology

Symptoms and signs

As mentioned earlier, hypertension does not typically cause symptoms unless it is very high, for example $> 200/120$ mmHg. **If very raised patients may experience:**

- headaches
- visual disturbance
- seizures

In terms of signs hypertension is obviously usually detected when checking someones blood pressure. For diagnosing longstanding blood pressure there has been a move in recent years to using 24 hour blood pressure monitors. These avoid cases of so called 'white coat' hypertension where a patients blood pressure rises when they are in a clinical setting, for example a GP surgery. Studies have shown that readings from 24 hour blood pressure monitors correlate better with clinical outcomes and hence should be used to guide decisions about treatment.

It also also important when assessing a patient with newly diagnosed hypertension to ensure they do not have any end-organ damage:

- fundoscopy: to check for hypertensive retinopathy
- urine dipstick: to check for renal disease, either as a cause or consequence of hypertension
- ECG: to check for left ventricular hypertrophy or ischaemic heart disease

Investigations

As mentioned previously 24 hour blood pressure is now recommend for the diagnosis of hypertension. If 24 hour blood pressure monitoring is not available then home readings using an automated sphygmomanometer are useful.

Following diagnosis patients typically have the following tests:

- urea and electrolytes: check for renal disease, either as a cause or consequence of hypertension
- HbA1c: check for co-existing diabetes mellitus, another important risk factor for cardiovascular disease
- lipids: check for hyperlipidaemia, again another important risk factor for cardiovascular disease
- ECG
- urine dipstick

Management:

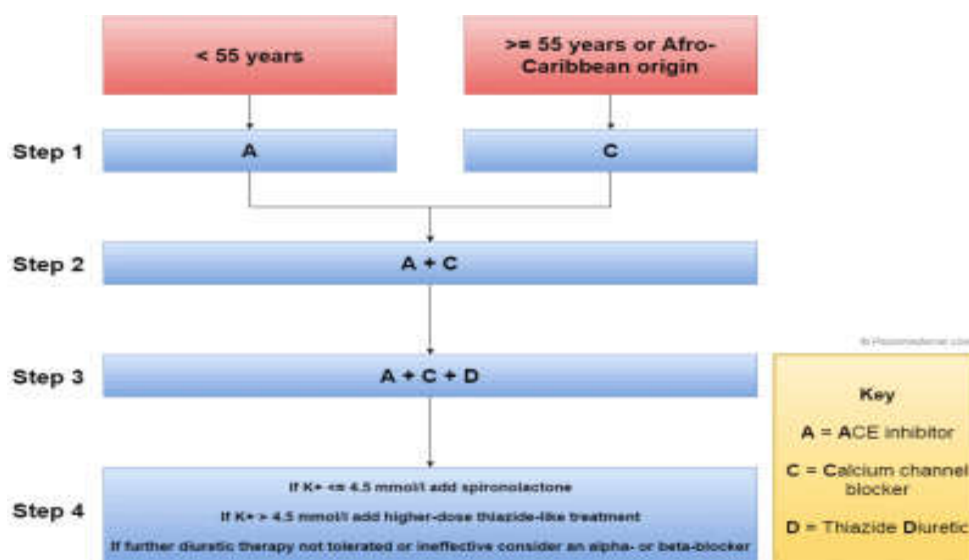
The management of patients with hypertension involves several aspects:

- drug therapy using antihypertensives
- modification of other risk factors to reduce the overall risk of cardiovascular disease
- monitoring the patient for the development of complications of hypertension.

The table below shows the common drugs used to treat hypertension:

Drug	Mechanism of action	Common side-effects	Notes
Angiotensin-converting enzyme (ACE) inhibitors	Inhibit the conversion angiotensin I to angiotensin II	Cough Angioedema Hyperkalaemia	First-line treatment in younger patients (< 55 years old) Less effective in Afro-Caribbean patients Must be avoided in pregnant women Renal function must be check 2-3 weeks after starting due to the risk of worsening renal function in patients with renovascular disease Drug names end in '-pril'
Calcium channel blockers	Block voltage-gated calcium channels relaxing vascular smooth muscle and force of myocardial contraction	Flushing Ankle swelling Headache	First-line treatment in older patients ($\geq$ 55 years old)
Thiazide type diuretics	Inhibit sodium absorption at the beginning of the distal convoluted tubule	Hyponatraemia Hypokalaemia Dehydration	Although technically a diuretic, thiazides have a very weak diuretic action
Angiotensin II receptor blockers (A2RB)	Block effects of angiotensin II at the AT1 receptor	Hyperkalaemia	Angiotensin II receptor blockers are generally used in situations where patients have not tolerated an ACE inhibitor, usually due to the development of a cough Drug names end in '-sartan'

Drug therapy is decided by well established NICE guidelines, which advocate a step-wise approach

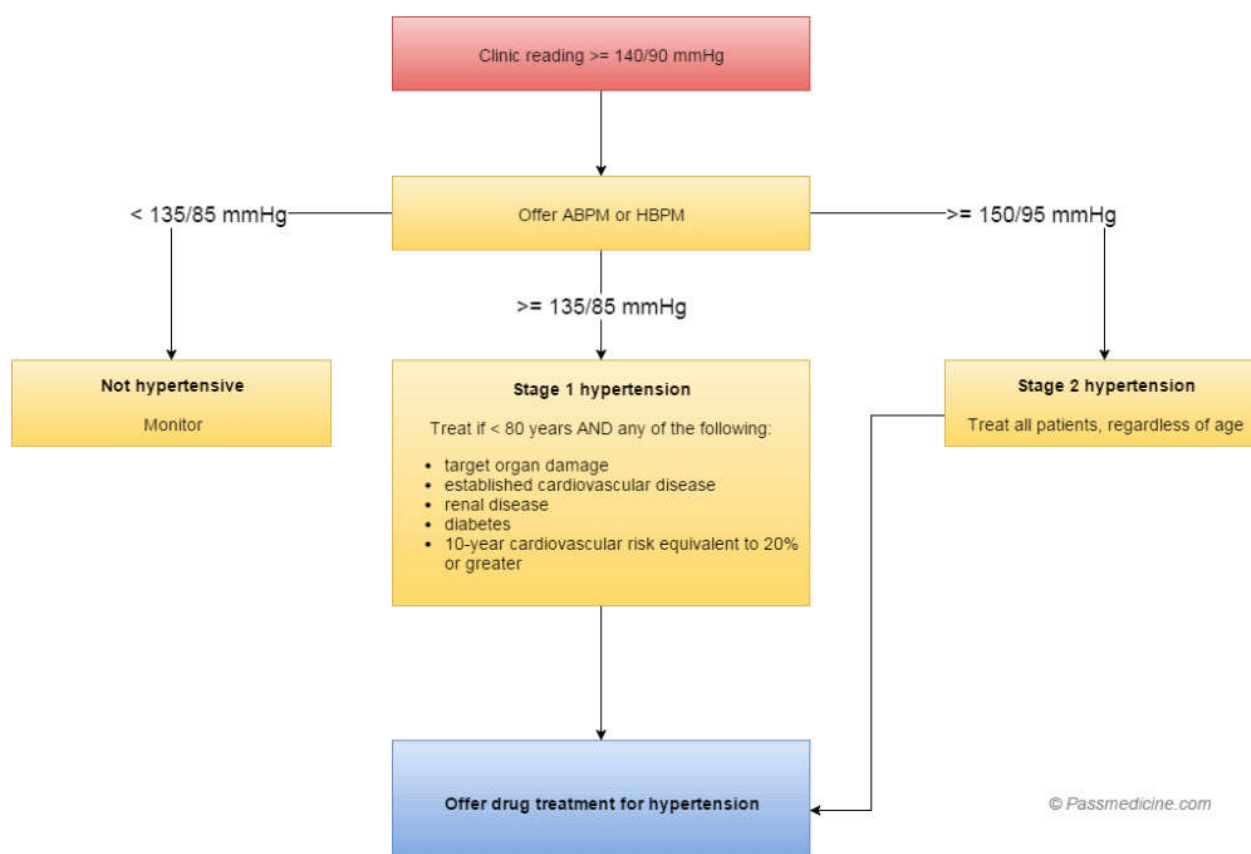


Flow chart showing the management of hypertension as per current NICE guidelines

Hypertension: diagnosis

NICE published updated guidelines for the management of hypertension in 2011. Some of the key changes include:

- classifying hypertension into stages
- recommending the use of ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM)



Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines.

Why were these guidelines needed?

It has long been recognised by doctors that there is a subgroup of patients whose blood pressure climbs 20 mmHg whenever they enter a clinical setting, so called 'white coat hypertension'. If we just rely on clinic readings then such patients may be diagnosed as having hypertension when, the vast majority of the time, their blood pressure is normal.

This has led to the use of both ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM) to confirm the diagnosis of hypertension. These techniques allow a more accurate assessment of a patients' overall blood pressure. Not only does this help prevent overdiagnosis of hypertension - ABPM has been shown to be a more accurate predictor of cardiovascular events than clinic readings.

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocate.

Stage	Criteria
Stage 1 hypertension	Clinic BP $\geq$ 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP $\geq$ 135/85 mmHg
Stage 2 hypertension	Clinic BP $\geq$ 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP $\geq$ 150/95 mmHg
Severe hypertension	Clinic systolic BP $\geq$ 180 mmHg, or clinic diastolic BP $\geq$ 110 mmHg

Diagnosing hypertension

- ◆ Firstly, NICE recommend measuring blood pressure in both arms when considering a diagnosis of hypertension.
- ◆ If the difference in readings between arms is more than 20 mmHg then the measurements should be repeated. If the difference remains > 20 mmHg then subsequent blood pressures should be recorded from the arm with the higher reading.
- ◆ It should of course be remembered that there are pathological causes of unequal blood pressure readings from the arms, such as supraventricular aortic stenosis. It is therefore prudent to listen to the heart sounds if a difference exists and further investigation if a very large difference is noted.
- ◆ NICE also recommend taking a second reading during the consultation, if the first reading is $> 140/90$ mmHg. The lower reading of the two should determine further management.
- ◆ NICE suggest offering ABPM or HBPM to any patient with a blood pressure $\geq 140/90$ mmHg.

If however the blood pressure is $\geq 180/110$ mmHg:

- immediate treatment should be considered
- if there are signs of papilloedema or retinal haemorrhages NICE recommend same day assessment by a specialist
- NICE also recommend referral if a pheochromocytoma is suspected (labile or postural hypotension, headache, palpitations, pallor and diaphoresis).

Ambulatory blood pressure monitoring (ABPM):

- at least 2 measurements per hour during the person's usual waking hours (for example, between 08:00 and 22:00)
- use the average value of at least 14 measurements

If ABPM is not tolerated or declined HBPM should be offered.

Home blood pressure monitoring (HBPM):

- for each BP recording, two consecutive measurements need to be taken, at least 1 minute apart and with the person seated
- BP should be recorded twice daily, ideally in the morning and evening
- BP should be recorded for at least 4 days, ideally for 7 days
- discard the measurements taken on the first day and use the average value of all the remaining measurements

Interpreting the results

ABPM/HBPM $\geq$ 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 20% or greater

ABPM/HBPM $\geq$ 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

External Links:

[NICE](#)

2011 Hypertension guidelines

Hypertension: management

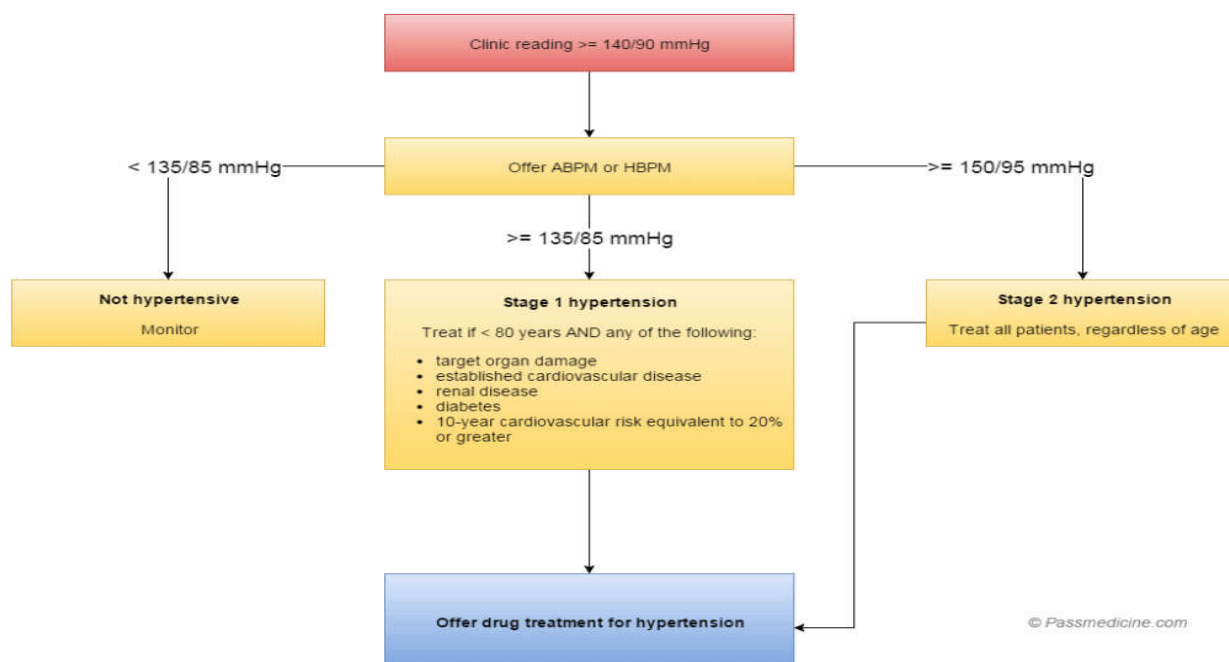
NICE published updated guidelines for the management of hypertension in 2011. Some of the key changes include:

- classifying hypertension into stages
- recommending the use of ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM)
- calcium channel blockers are now considered superior to thiazides
- bendroflumethiazide is no longer the thiazide of choice

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocate.

Stage	Criteria
Stage 1 hypertension	Clinic BP $\geq 140/90$ mmHg and subsequent ABPM daytime average or HBPM average BP $\geq 135/85$ mmHg
Stage 2 hypertension	Clinic BP $\geq 160/100$ mmHg and subsequent ABPM daytime average or HBPM average BP $\geq 150/95$ mmHg
Severe hypertension	Clinic systolic BP ≥ 180 mmHg, or clinic diastolic BP ≥ 110 mmHg



Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

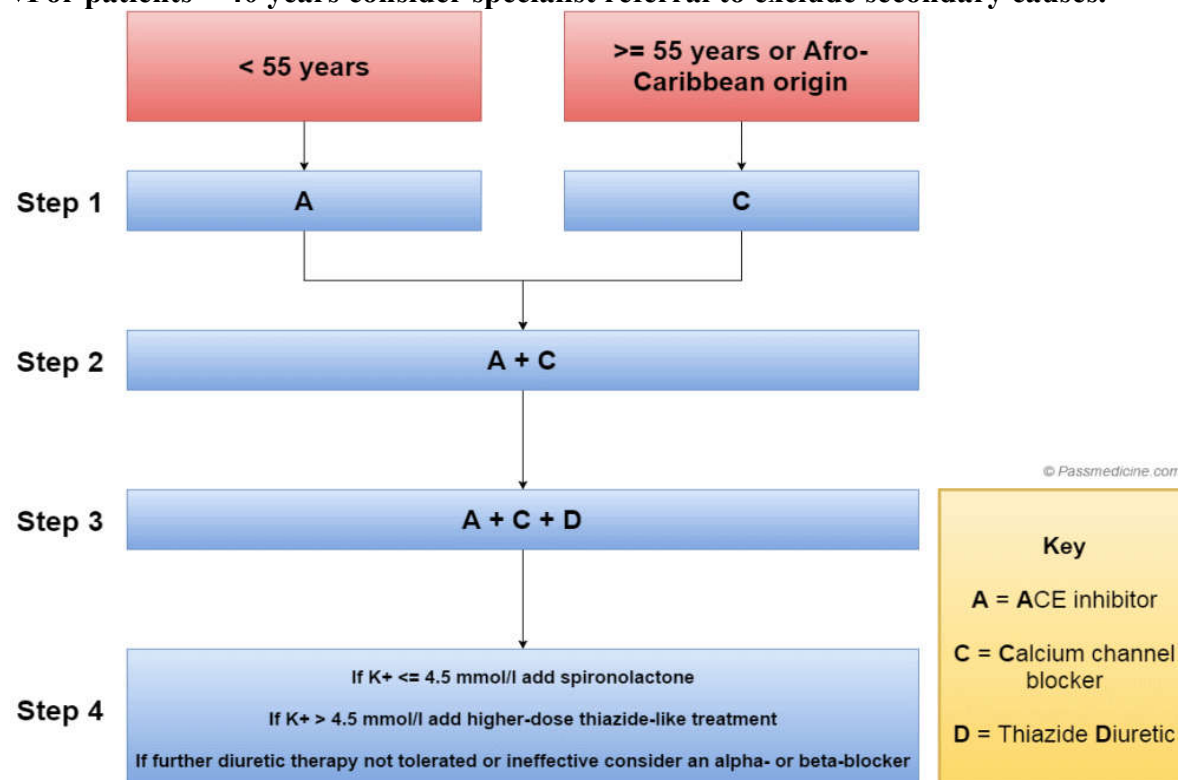
ABPM/HBPM $\geq$ 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 20% or greater

ABPM/HBPM $\geq$ 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

♦For patients < 40 years consider specialist referral to exclude secondary causes.



Flow chart showing the management of hypertension as per current NICE guidelines

Chapter: cardiology

Step 1 treatment:

- patients < 55-years-old: ACE inhibitor (A)
- patients $\geq$ 55-years-old or of Afro-Caribbean origin: calcium channel blocker

Step 2 treatment:

- ACE inhibitor + calcium channel blocker (A + C)

Step 3 treatment

- add a thiazide diuretic (D, i.e. A + C + D)
- NICE now advocate using either chlorthalidone (12.5-25.0 mg once daily) or indapamide (1.5 mg modified-release once daily or 2.5 mg once daily) in preference to a conventional thiazide diuretic such as bendroflumethiazide

NICE define a clinic BP $\geq$ 140/90 mmHg after step 3 treatment with optimal or best tolerated doses as resistant hypertension. They suggest step 4 treatment or seeking expert advice

Step 4 treatment:

- consider further diuretic treatment
- if potassium < 4.5 mmol/l add spironolactone 25mg od
- if potassium > 4.5 mmol/l add higher-dose thiazide-like diuretic treatment
- if further diuretic therapy is not tolerated, or is contraindicated or ineffective, consider an alpha- or beta-blocker

♦Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.

Blood pressure targets

	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

New drugs

Direct renin inhibitors:

- e.g. Aliskiren (branded as Rasilez)
- by inhibiting renin blocks the conversion of angiotensinogen to angiotensin I
- no trials have looked at mortality data yet. Trials have only investigated fall in blood pressure. Initial trials suggest aliskiren reduces blood pressure to a similar extent as angiotensin converting enzyme (ACE) inhibitors or angiotensin-II receptor antagonists
- adverse effects were uncommon in trials although diarrhoea was occasionally seen
- only current role would seem to be in patients who are intolerant of more established antihypertensive drugs

*BMJ 2013;346:f1325

External Links:

[NICE](#)

2011 Hypertension guidelines

Hypertension: secondary causes

It is thought that between 5-10% of patients diagnosed with hypertension have **primary hyperaldosteronism**, including Conn's syndrome. This makes it the single most common cause of secondary hypertension.

Renal disease accounts for a large percentage of the other cases of secondary hypertension. Conditions which may increase the blood pressure include:

- glomerulonephritis
- pyelonephritis
- adult polycystic kidney disease
- renal artery stenosis.

Endocrine disorders (other than primary hyperaldosteronism) may also result in increased blood pressure:

- phaeochromocytoma
- Cushing's syndrome
- Liddle's syndrome
- congenital adrenal hyperplasia (11-beta hydroxylase deficiency)
- acromegaly

Other causes include:

- NSAIDs
- pregnancy
- coarctation of the aorta
- the combined oral contraceptive pill
- steroids
- MAOI

Implantable cardiac defibrillators

Indications

- long QT syndrome
 - hypertrophic obstructive cardiomyopathy
 - previous cardiac arrest due to VT/VF
 - previous myocardial infarction with non-sustained VT on 24 hr monitoring, inducible VT on electrophysiology testing and ejection fraction < 35%
 - Brugada syndrome
-

Infective endocarditis

The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis. The following types of patients are affected:

- previously normal valves (50%, typically acute presentation)
- rheumatic valve disease (30%)
- prosthetic valves
- congenital heart defects
- intravenous drug users (IVDUs, e.g. typically causing tricuspid lesion)

Causes

- historically *Streptococcus viridans* was the most common cause of infective endocarditis. This is no longer the case, except in developing countries. ***Staphylococcus aureus* is now the most common cause of infective endocarditis.** *Staphylococcus aureus* is also particularly common in acute presentation and IVDUs
- coagulase-negative Staphylococci such as *Staphylococcus epidermidis* commonly colonize indwelling lines and are the most cause of endocarditis in patients following prosthetic valve surgery, usually the result of perioperative contamination. After 2 months the spectrum of organisms which cause endocarditis return to normal (i.e. *Staphylococcus aureus* is the most common cause)
- *Streptococcus viridans* still accounts for around 20% of cases. Technically *Streptococcus viridans* is a pseudotaxonomic term, referring to viridans streptococci, rather than a particular organism. The two most notable viridans streptococci are *Streptococcus mitis* and *Streptococcus sanguinis*. They are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure
- *Streptococcus bovis* is associated with colorectal cancer
- non-infective: systemic lupus erythematosus (Libman-Sacks), malignancy: marantic endocarditis

Culture negative causes:

- prior antibiotic therapy
- *Coxiella burnetii*
- *Bartonella*
- *Brucella*
- HACEK: *Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Eikenella*, *Kingella*)

*Lancet 2016; 387: 882-93 Infective Endocarditis

External Links

[American Heart Association](#)

Infective endocarditis guidelines

[NICE](#)

2008 Prophylaxis against infective endocarditis

Infective endocarditis: Modified Duke criteria

Infective endocarditis diagnosed if:

- pathological criteria positive, or
- 2 major criteria, or
- 1 major and 3 minor criteria, or
- 5 minor criteria

Pathological criteria

Positive histology or microbiology of pathological material obtained at autopsy or cardiac surgery (valve tissue, vegetations, embolic fragments or intracardiac abscess content)

Major criteria

Positive blood cultures:

- two positive blood cultures showing typical organisms consistent with infective endocarditis, such as *Streptococcus viridans* and the HACEK group, or
- persistent bacteraemia from two blood cultures taken > 12 hours apart or three or more positive blood cultures where the pathogen is less specific such as *Staph aureus* and *Staph epidermidis*, or
- positive serology for *Coxiella burnetii*, *Bartonella* species or *Chlamydia psittaci*, or
- positive molecular assays for specific gene targets

Evidence of endocardial involvement:

- positive echocardiogram (oscillating structures, abscess formation, new valvular regurgitation or dehiscence of prosthetic valves), or
- new valvular regurgitation.

Minor criteria

- predisposing heart condition or intravenous drug use
- microbiological evidence does not meet major criteria
- fever $> 38^{\circ}\text{C}$
- vascular phenomena: major emboli, splenomegaly, clubbing, splinter haemorrhages, Janeway lesions, petechiae or purpura
- immunological phenomena: glomerulonephritis, Osler's nodes, Roth spots

External Links:

[European Society of Cardiology](#)

Infective endocarditis guidelines

Infective endocarditis: prophylaxis

The 2008 guidelines from NICE have radically changed the list of procedures for which antibiotic prophylaxis is recommended

NICE recommends the following procedures do not require prophylaxis:

- dental procedures
- upper and lower gastrointestinal tract procedures
- genitourinary tract; this includes urological, gynaecological and obstetric procedures and childbirth
- upper and lower respiratory tract; this includes ear, nose and throat procedures and bronchoscopy

The guidelines do however suggest:

- any episodes of infection in people at risk of infective endocarditis should be investigated and treated promptly to reduce the risk of endocarditis developing
- if a person at risk of infective endocarditis is receiving antimicrobial therapy because they are undergoing a gastrointestinal or genitourinary procedure at a site where there is a suspected infection they should be given an antibiotic that covers organisms that cause infective endocarditis

External Links

[NICE](#)

2008 Infective endocarditis prophylaxis guidelines

Isolated systolic hypertension

Isolated systolic hypertension (ISH) is common in the elderly, affecting around 50% of people older than 70 years old. The Systolic Hypertension in the Elderly Program (SHEP) back in 1991 established that treating ISH reduced both strokes and ischaemic heart disease. Drugs such as thiazides were recommended as first line agents. This approach is contradicted by the 2011 NICE guidelines which recommends treating ISH in the same stepwise fashion as standard hypertension.

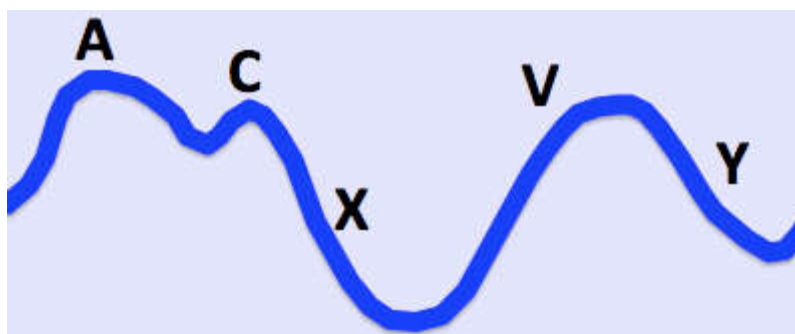
External Links:

[NICE](#)

2011 Hypertension guidelines.

Jugular venous pulse

As well as providing information on right atrial pressure, the jugular vein waveform may provide clues to underlying valvular disease. A non-pulsatile JVP is seen in superior vena caval obstruction. Kussmaul's sign describes a paradoxical rise in JVP during inspiration seen in constrictive pericarditis.



'a' wave = atrial contraction

- large if atrial pressure e.g. tricuspid stenosis, pulmonary stenosis, pulmonary hypertension
- absent if in atrial fibrillation

Cannon 'a' waves

- caused by atrial contractions against a closed tricuspid valve
- are seen in complete heart block, ventricular tachycardia/ectopics, nodal rhythm, single chamber ventricular pacing

'c' wave

- closure of tricuspid valve
- not normally visible

'v' wave

- due to passive filling of blood into the atrium against a closed tricuspid valve
- giant v waves in tricuspid regurgitation

'x' descent = fall in atrial pressure during ventricular systole

'y' descent = opening of tricuspid valve

JVP: cannon waves

Caused by the right atrium contracting against a closed tricuspid valve. May be subdivided into regular or intermittent

Regular cannon waves:

- ventricular tachycardia (with 1:1 ventricular-atrial conduction)
- atrio-ventricular nodal re-entry tachycardia (AVNRT)

Irregular cannon waves:

- complete heart block
-

Long QT syndrome

Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia and can therefore cause collapse/sudden death. The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. A normal corrected QT interval is less than 430 ms in males and 450 ms in females.

Causes of a prolonged QT interval:

Congenital	Drugs*	Other
♦Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) ♦Romano-Ward syndrome (no deafness)	♦ amiodarone, sotalol, class 1a antiarrhythmic drugs ♦tricyclic antidepressants, selective serotonin reuptake inhibitors (especially citalopram) ♦ methadone ♦ chloroquine ♦terfenadine** ♦ erythromycin ♦ haloperidol	♦electrolyte:hypocalcaemia,hypokalaemia,hypomagnesaemia ♦acute myocardial infarction ♦myocarditis ♦hypothermia ♦ subarachnoid haemorrhage

Features

- may be picked up on routine ECG or following family screening
- Long QT1 - usually associated with exertional syncope, often swimming
- Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli
- Long QT3 - events often occur at night or at rest
- sudden cardiac death

Management

- avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g. Strenuous exercise)
- beta-blockers***
- implantable cardioverter defibrillators in high risk cases

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels. See the link for more details

**a non-sedating antihistamine and classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time

***note sotalol may exacerbate long QT syndrome.

External Links:

[Life in the Fast Lane](#)

QT Interval and Long QT syndrome

[Pharmacology Review](#)

2010 Drug-Induced Long QT syndrome

Malignant hypertension

Basics

- severe hypertension (e.g. >200/130 mmHg)
- occurs in both essential and secondary types
- fibrinoid necrosis of blood vessels, leading to retinal haemorrhages, exudates, and proteinuria, haematuria due to renal damage (benign nephrosclerosis).
- can lead to cerebral oedema → encephalopathy

Features

- classically: severe headaches, nausea/vomiting, visual disturbance
- however chest pain and dyspnoea common presenting symptoms
- papilloedema
- severe: encephalopathy (e.g. seizures)

Management

- reduce diastolic no lower than 100mmHg within 12-24 hrs
- bed rest
- most patients: oral therapy e.g. atenolol
- if severe/encephalopathic: IV sodium nitroprusside/labetolol.

Mitral regurgitation

Features

- pan-systolic murmur
 - soft S1, split S2
-

Mitral stenosis

It is said that the causes of mitral stenosis are rheumatic fever, rheumatic fever and rheumatic fever. Rarer causes that may be seen in the exam include mucopolysaccharidoses, carcinoid and endocardial fibroelastosis

Features

- mid-late diastolic murmur (best heard in expiration)
- loud S1, opening snap
- low volume pulse
- malar flush
- atrial fibrillation.

Features of severe MS

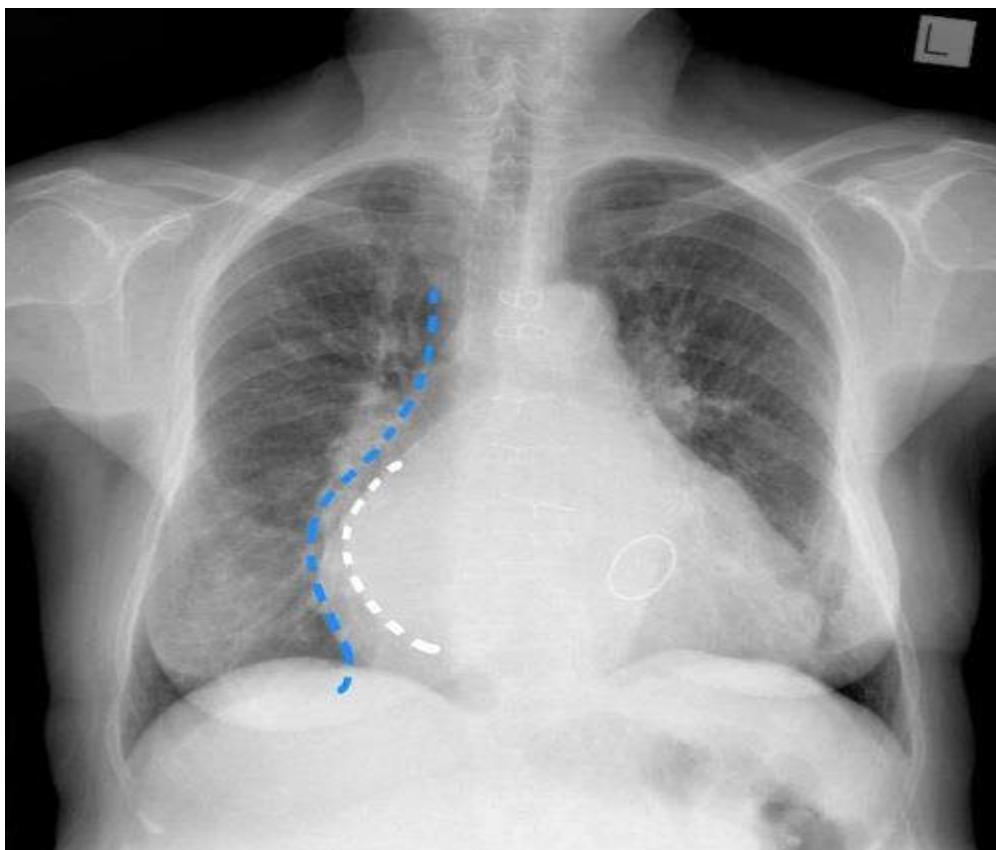
- length of murmur increases
- opening snap becomes closer to S2

Chest x-ray

- left atrial enlargement may be seen

Echocardiography

- the normal cross sectional area of the mitral valve is 4-6 sq cm. A 'tight' mitral stenosis implies a cross sectional area of < 1 sq cm.



© Image used on license from [Radiopaedia](#)

Chest x-ray from a patient with mitral stenosis. This patient has had a sternotomy and a prosthetic mitral valve. There is splaying of the carina with elevation of the left main bronchus, a double right heart border and cardiomegaly. The features are those of left atrial enlargement. Although the entire heart is enlarged, a double contour is seen through the right side of the heart. The more medial line is the enlarged left atrium (white dotted line) and the heart heart border is more lateral (blue dotted line).

Mitral valve prolapse

Mitral valve prolapse is common, occurring in around 5-10 % of the population. It is usually idiopathic but may be associated with a wide variety of cardiovascular disease and other conditions

Associations:

- congenital heart disease: PDA, ASD
- cardiomyopathy
- Turner's syndrome
- Marfan's syndrome, Fragile X
- osteogenesis imperfecta

Chapter: cardiology

- pseudoxanthoma elasticum
- Wolff-Parkinson White syndrome
- long-QT syndrome
- Ehlers-Danlos Syndrome
- polycystic kidney disease

Features:

- patients may complain of atypical chest pain or palpitations
- mid-systolic click (occurs later if patient squatting)
- late systolic murmur (longer if patient standing)
- complications: mitral regurgitation, arrhythmias (including long QT), emboli, sudden death

Multifocal atrial tachycardia

Multifocal atrial tachycardia (MAT) may be defined as an irregular cardiac rhythm caused by at least three different sites in the atria, which may be demonstrated by morphologically distinctive P waves. It is more common in elderly patients with chronic lung disease, for example COPD

Management

- correction of hypoxia and electrolyte disturbances
- rate-limiting calcium channel blockers are often used first-line
- cardioversion and digoxin are not useful in the management of MAT

External Links

[Medscape](#)

Multifocal atrial tachycardia management.

Murmurs

Ejection systolic:

- aortic stenosis
- pulmonary stenosis, HOCM
- ASD, Fallot's

Holosystolic (pansystolic)

- mitral/tricuspid regurgitation (high-pitched and 'blowing' in character)
- VSD ('harsh' in character)

Late systolic

- mitral valve prolapse
- coarctation of aorta

Early diastolic:

- aortic regurgitation (high-pitched and 'blowing' in character)
- Graham-Steel murmur (pulmonary regurgitation, again high-pitched and 'blowing' in character)

Mid-late diastolic:

- mitral stenosis ('rumbling' in character)
- Austin-Flint murmur (severe aortic regurgitation, again is 'rumbling' in character)

Continuous machine-like mumur:

- patent ductus arteriosus

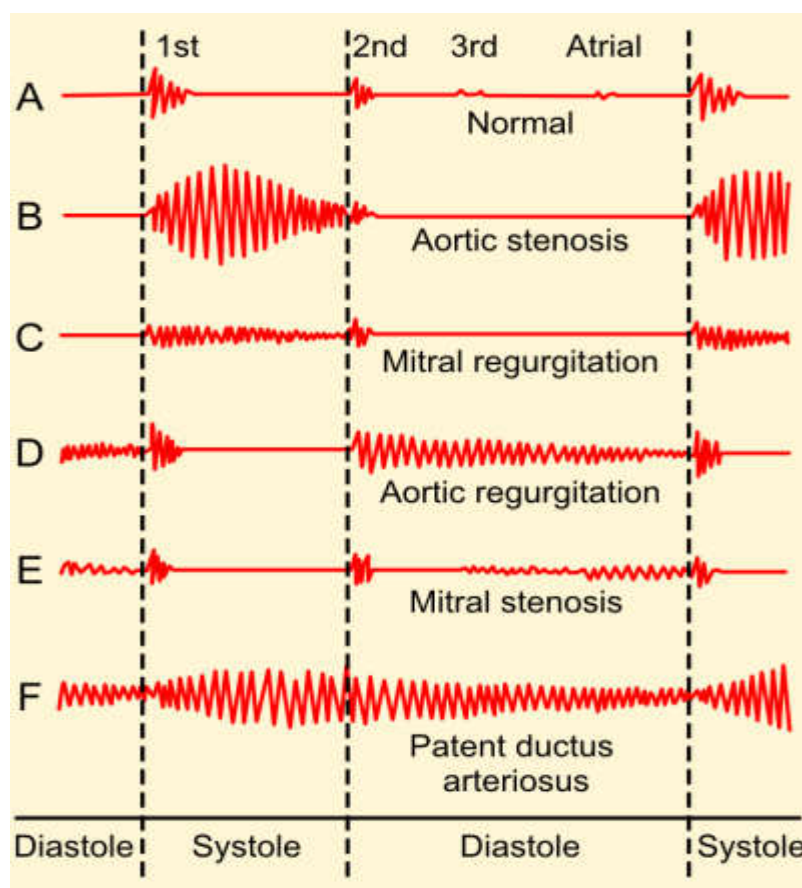


Image sourced from [Wikipedia](#)

External Links

[University of Washington](#)

Audio clips of common murmurs / heart sounds

Myocardial infarction: complications

Patients are at risk of a number of immediate, early and late complications following a myocardial infarction (MI).

Cardiac arrest

This most commonly occurs due to patients developing ventricular fibrillation and is the most common cause of death following a MI. Patients are managed as per the ALS protocol with defibrillation.

Cardiogenic shock

If a large part of the ventricular myocardium is damaged in the infarction the ejection fraction of the heart may decrease to the point that the patient develops cardiogenic shock. This is difficult to treat. Other causes of cardiogenic shock include the 'mechanical' complications such as left ventricular free wall rupture as listed below. Patients may require inotropic support and/or an intra-aortic balloon pump.

Chronic heart failure

As described above, if the patient survives the acute phase their ventricular myocardium may be dysfunctional resulting in chronic heart failure. Loop diuretics such as furosemide will decrease fluid overload. Both ACE-inhibitors and beta-blockers have been shown to improve the long-term prognosis of patients with chronic heart failure.

Tachyarrhythmias

Ventricular fibrillation, as mentioned above, is the most common cause of death following a MI. Other common arrhythmias including ventricular tachycardia.

Bradyarrhythmias

Atrioventricular block is more common following inferior myocardial infarctions.

Pericarditis

Pericarditis in the first 48 hours following a transmural MI is common (c. 10% of patients). The pain is typical for pericarditis (worse on lying flat etc), a pericardial rub may be heard and a pericardial effusion may be demonstrated with an echocardiogram.

Dressler's syndrome tends to occur around 2-6 weeks following a MI. The underlying pathophysiology is thought to be an autoimmune reaction against antigenic proteins formed as the myocardium recovers. It is characterised by a combination of fever, pleuritic pain, pericardial effusion and a raised ESR. It is treated with NSAIDs.

Left ventricular aneurysm

The ischaemic damage sustained may weaken the myocardium resulting in aneurysm formation. This is typically associated with persistent ST elevation and left ventricular failure. Thrombus may form within the aneurysm increasing the risk of stroke. Patients are therefore anticoagulated.

Left ventricular free wall rupture

This is seen in around 3% of MIs and occurs around 1-2 weeks afterwards. Patients present with acute heart failure secondary to cardiac tamponade (raised JVP, pulsus paradoxus, diminished heart sounds). Urgent pericardiocentesis and thoracotomy are required.

Ventricular septal defect

Rupture of the interventricular septum usually occurs in the first week and is seen in around 1-2% of patients. Features: acute heart failure associated with a pan-systolic murmur. An echocardiogram is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion. Urgent surgical correction is needed.

Acute mitral regurgitation

More common with infero-posterior infarction and may be due to ischaemia or rupture of the papillary muscle. An early-to-mid systolic murmur is typically heard. Patients are treated with vasodilator therapy but often require emergency surgical repair.

Myocardial infarction: secondary prevention

NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2013. Some key points are listed below

All patients should be offered the following drugs:

- dual antiplatelet therapy (aspirin plus a second antiplatelet agent)
- ACE inhibitor
- beta-blocker
- statin

Some selected lifestyle points:

- diet: advise a Mediterranean style diet, switch butter and cheese for plant oil based products. Do not recommend omega-3 supplements or eating oily fish
- exercise: advise 20-30 mins a day until patients are 'slightly breathless'
- sexual activity may resume 4 weeks after an uncomplicated MI. Reassure patients that sex does not increase their likelihood of a further MI. PDE5 inhibitors (e.g, sildenafil) may be used 6 months after a MI. They should however be avoided in patient prescribed either nitrates or nicorandil

Clopidogrel

- since clopidogrel came off patent it is now much more widely used post-MI
- STEMI: the European Society of Cardiology recommend dual antiplatelets for 12 months. In the UK this means aspirin + clopidogrel
- non-ST segment elevation myocardial infarction (NSTEMI): following the NICE 2013 *Secondary prevention in primary and secondary care for patients following a myocardial infarction* guidelines clopidogrel should be given for the first 12 months.

Aldosterone antagonists

- patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI treatment (e.g. eplerenone) should be initiated within 3-14 days of the MI, preferably after ACE inhibitor therapy

External Links:

[NICE](#)

2013 Myocardial infarction: cardiac rehabilitation and prevention of further cardiovascular disease

[NICE](#)

2010 Unstable angina and NSTEMI guidelines

[European Society of Cardiology](#)

2012 STEMI guidelines

Myocardial infarction: STEMI management

A number of studies over the past 10 years have provided an evidence for the management of ST-elevation myocardial infarction (STEMI)

In the absence of contraindications, all patients should be given:

- **aspirin**
- **P2Y12-receptor antagonist.** Clopidogrel was the first P2Y12-receptor antagonist to be widely used but now ticagrelor is often favoured as studies have shown improved outcomes compared to clopidogrel, but at the expense of slightly higher rates of bleeding. This approach is supported in SIGN's 2016 guidelines. They also recommend that prasugrel (another P2Y12-receptor antagonist) could be considered if the patient is going to have a percutaneous coronary intervention
- **unfractionated heparin** is usually given for patients who're are going to have a PCI. Alternatives include low-molecular weight heparin.

NICE suggest the following in terms of oxygen therapy:

- do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:
- people with oxygen saturation (SpO₂) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO₂ of 94-98%.
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO₂ of 88-92% until blood gas analysis is available.

Primary percutaneous coronary intervention (PCI) has emerged as the gold-standard treatment for STEMI but is not available in all centres. Thrombolysis should be performed in patients without access to primary PCI.

With regards to thrombolysis:

- tissue plasminogen activator (tPA) has been shown to offer clear mortality benefits over streptokinase
- tenecteplase is easier to administer and has been shown to have non-inferior efficacy to alteplase with a similar adverse effect profile

An ECG should be performed 90 minutes following thrombolysis to assess whether there has been a greater than 50% resolution in the ST elevation

- if there has not been adequate resolution then rescue PCI is superior to repeat thrombolysis
- for patients successfully treated with thrombolysis PCI has been shown to be beneficial. The optimal timing of this is still under investigation

Glycaemic control in patients with diabetes mellitus

- in 2011 NICE issued guidance on the management of hyperglycaemia in acute coronary syndromes
- it recommends using a dose-adjusted insulin infusion with regular monitoring of blood glucose levels to glucose below 11.0 mmol/l
- intensive insulin therapy (an intravenous infusion of insulin and glucose with or without potassium, sometimes referred to as 'DIGAMI') regimes are not recommended routinely

External Links

[NICE](#)

2010 Chest pain of recent onset guidelines

[NICE](#)

2011 Managing hyperglycaemia in acute coronary syndromes

[SIGN](#)

2016 Acute coronary syndrome guidelines.

Myocarditis

Causes

- viral: coxsackie, HIV
- bacteria: diphtheria, clostridia
- spirochaetes: Lyme disease
- protozoa: Chagas' disease, toxoplasmosis
- autoimmune
- drugs: doxorubicin

Presentation

- usually young patient with acute history
- chest pain, SOB

Nitrates

Nitrates are a group of drugs which have vasodilating effects. The main indications for their use is in the management of angina and the acute treatment of heart failure. Sublingual glyceryl trinitrate is the most common drug used in patients with ischaemic heart disease to relieve angina attacks.

Mechanism of action:

- cause release of nitric oxide in smooth muscle, increasing cGMP which leads to a fall in intracellular calcium levels
- in angina they both dilate the coronary arteries and also reduce venous return which in turn reduces left ventricular work, reducing myocardial oxygen demand.

Side-effects:

- hypotension
- tachycardia
- headaches
- flushing.

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- the BNF advises that patients who develop tolerance should take the second dose of isosorbide mononitrate after 8 hours, rather than after 12 hours. This allows blood-nitrate levels to fall for 4 hours and maintains effectiveness
- this effect is not seen in patients who take modified release isosorbide mononitrate

Overdose and poisoning: management

The table below outlines the main management for common overdoses:

Toxin	Treatment
Paracetamol	Management • activated charcoal if ingested < 1 hour ago • N-acetylcysteine (NAC) • liver transplantation
Salicylate	Management • urinary alkalinization is now rarely used - it is contraindicated in cerebral and pulmonary edema with most units now proceeding straight to haemodialysis in cases of severe poisoning • haemodialysis
Opioid/opiates	Naloxone
Benzodiazepines	Flumazenil
Tricyclic antidepressants	Management IV bicarbonate may reduce the risk of seizures and arrhythmias in severe toxicity arrhythmias: class 1a (e.g. Quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation. Class III drugs such as amiodarone should also be avoided as they prolong the QT interval. Response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in management of tricyclic induced arrhythmias • dialysis is ineffective in removing tricyclics.

Lithium	Management • mild-moderate toxicity may respond to volume resuscitation with normal saline • haemodialysis may be needed in severe toxicity • sodium bicarbonate is sometimes used but there is limited evidence to support this. By increasing the alkalinity of the urine it promotes lithium excretion
Warfarin	Vitamin K, prothrombin complex
Heparin	Protamine sulphate
Beta-blockers	Management • if bradycardic then atropine • in resistant cases glucagon may be used
Ethylene glycol	Management has changed in recent times • ethanol has been used for many years • works by competing with ethylene glycol for the enzyme alcohol dehydrogenase • this limits the formation of toxic metabolites (e.g. Glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning • fomepizole, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol • haemodialysis also has a role in refractory cases
Methanol poisoning	Management • fomepizole or ethanol • haemodialysis
Organophosphate insecticides	Management • atropine • the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit

Chapter: cardiology

Digoxin	Digoxin-specific antibody fragments
Iron	Desferrioxamine, a chelating agent
Lead	Dimercaprol, calcium edetate
Carbon monoxide	Management • 100% oxygen • hyperbaric oxygen
Cyanide	Hydroxocobalamin; also combination of amyl nitrite, sodium nitrite, and sodium thiosulfate

External Links:

[Royal College of Physicians](#)

2012 Poisoning and self-harm update

[Royal College of Physicians](#)

2007 Acute poisoning on the medical admissions unit

Pacemakers: temporary

Indications for a temporary pacemaker

- symptomatic/haemodynamically unstable bradycardia, not responding to atropine
- post-ANTERIOR MI: type 2 or complete heart block*
- trifascicular block prior to surgery

*post-INFERIOR MI complete heart block is common and can be managed conservatively if asymptomatic and haemodynamically stable

Paradoxical embolisation

For a right-sided thrombus (e.g. DVT) to cause a left-sided embolism (e.g. stroke) it must obviously pass from the right-to-left side of the heart

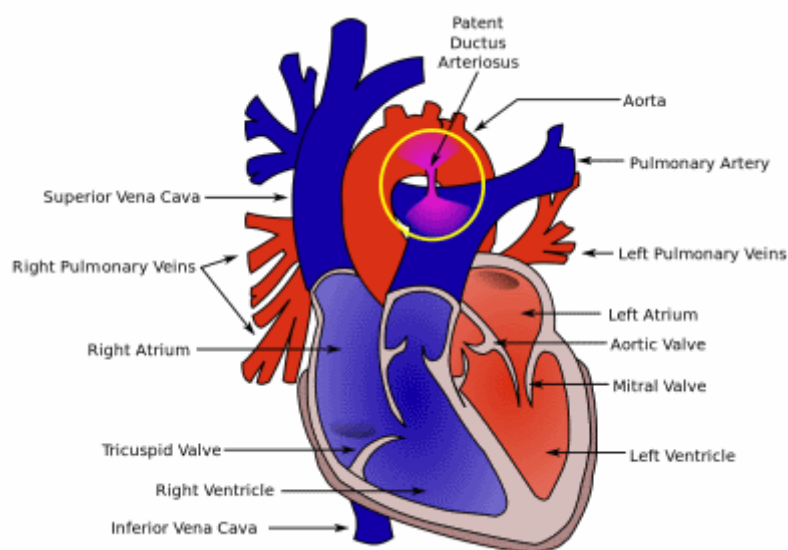
The following cardiac lesions may cause such events;

- patent foramen ovale - present in around 20% of the population
- atrial septal defect - a much less common cause

Patent ductus arteriosus

Overview

- acyanotic congenital heart defect
- connection between the pulmonary trunk and descending aorta
- more common in premature babies, born at high altitude or maternal rubella infection in the first trimester



Features

- left subclavicular thrill
- continuous 'machinery' murmur
- large volume, bounding, collapsing pulse
- wide pulse pressure
- heaving apex beat

Management

- indomethacin closes the connection in the majority of cases
- if associated with another congenital heart defect amenable to surgery then prostaglandin E1 is useful to keep the duct open until after surgical repair

Patent foramen ovale

Patent foramen ovale (PFO) is present in around 20% of the population. It may allow embolus (e.g. from DVT) to pass from right side of the heart to the left side leading to a stroke - 'a paradoxical embolus'

There also appears to be an association between migraine and PFO. Some studies have reported improvement in migraine symptoms following closure of the PFO

Percutaneous coronary intervention

Percutaneous coronary intervention (PCI) is a technique used to restore myocardial perfusion in patients with ischaemic heart disease, both in patients with stable angina and acute coronary syndromes. Stents are implanted in around 95% of patients - it is now rare for just balloon angioplasty to be performed

Following stent insertion migration and proliferation of smooth muscle cells and fibroblasts occur to the treated segment. The stent struts eventually become covered by endothelium. Until this happens there is an increased risk of platelet aggregation leading to thrombosis.

Two main complications may occur:

- stent thrombosis: due to platelet aggregation as above. Occurs in 1-2% of patients, most commonly in the first month. Usually presents with acute myocardial infarction
- restenosis: due to excessive tissue proliferation around stent. Occurs in around 5-20% of patients, most commonly in the first 3-6 months. Usually presents with the recurrence of angina symptoms. Risk factors include diabetes, renal impairment and stents in venous bypass grafts

Types of stent

- bare-metal stent (BMS)
- drug-eluting stents (DES): stent coated with paclitaxel or rapamycin which inhibit local tissue growth. Whilst this reduces restenosis rates the stent thrombosis rates are increased as the process of stent endothelialisation is slowed

Following insertion the most important factor in preventing stent thrombosis is antiplatelet therapy. Aspirin should be continued indefinitely. The length of clopidogrel treatment depends on the type of stent, reason for insertion and consultant preference.

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Chapter: cardiology

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Peri-arrest rhythms: bradycardia

The 2015 Resuscitation Council (UK) guidelines emphasise that the management of bradycardia depends on:

1. identifying the presence of signs indicating haemodynamic compromise - 'adverse signs'
2. identifying the potential risk of asystole

Adverse signs

The following factors indicate haemodynamic compromise and hence the need for treatment:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

Atropine is the first line treatment in this situation. If this fails to work, or there is the potential risk of asystole then transvenous pacing is indicated.

Potential risk of asystole

The following indicate a potential risk of asystole and hence the need for treatment with transvenous pacing:

- complete heart block with broad complex QRS
- recent asystole
- Mobitz type II AV block
- ventricular pause > 3 seconds

If there is a delay in the provision of transvenous pacing the following interventions may be used:

- atropine, up to maximum of 3mg
- transcutaneous pacing
- adrenaline infusion titrated to response

External Links

[Resuscitation Council](#)

2015 peri-arrest guidelines

Peri-arrest rhythms: tachycardia

The 2015 Resuscitation Council (UK) guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment algorithm can be found at the Resuscitation Council website, below is a very limited summary:

Broad-complex tachycardia

Regular

- assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
- loading dose of amiodarone followed by 24 hour infusion.
-

Irregular

1. AF with bundle branch block - treat as for narrow complex tachycardia
2. Polymorphic VT (e.g. Torsade de pointes) - IV magnesium

Narrow-complex tachycardia

Regular

- vagal manoeuvres followed by IV adenosine
- if above unsuccessful consider diagnosis of atrial flutter and control rate (e.g. Beta-blockers)

Irregular

- probable atrial fibrillation
- if onset < 48 hr consider electrical or chemical cardioversion
- rate control (e.g. Beta-blocker or digoxin) and anticoagulation

External Links

[Resuscitation Council](#)

2015 peri-arrest guidelines

Primary pulmonary hypertension

The classification of pulmonary hypertension is currently changing with the term idiopathic pulmonary arterial hypertension (IPAH) becoming more widely used

Primary pulmonary hypertension (PPH, now IPAH)

- pulmonary arterial pressure > 25 mmHg at rest, > 30mmHg with exercise
- PPH is diagnosed when no underlying cause can be found
- around 10% of cases are familial: autosomal dominant
- endothelin thought to play a key role in pathogenesis
- associated with HIV, cocaine and anorexigens (e.g. fenfluramine).

Chapter: cardiology

Features

- more common in females, typically presents at 20-40 years old
- progressive SOB
- cyanosis.
- right ventricular heave, loud P2, raised JVP with prominent 'a' waves, tricuspid regurgitation.

Pulses

Pulsus paradoxus

- greater than the normal (10 mmHg) fall in systolic blood pressure during inspiration → faint or absent pulse in inspiration
- severe asthma, cardiac tamponade

Slow-rising/plateau

- aortic stenosis

Collapsing

- aortic regurgitation
- patent ductus arteriosus
- hyperkinetic (anaemia, thyrotoxic, fever, exercise/pregnancy)

Pulsus alternans

- regular alternation of the force of the arterial pulse
- severe LVF

Bisferiens pulse

- 'double pulse' - two systolic peaks
- mixed aortic valve disease

'Jerky' pulse

- hypertrophic obstructive cardiomyopathy*

*HOCM may occasionally be associated with a bisferiens pulse

Restrictive cardiomyopathy

Features

- similar to constrictive pericarditis

Features suggesting restrictive cardiomyopathy rather than constrictive pericarditis:

- prominent apical pulse
- absence of pericardial calcification on CXR
- heart may be enlarged
- ECG abnormalities e.g. bundle branch block, Q waves

Causes

- amyloidosis (e.g. secondary to myeloma) - most common cause in UK
- haemochromatosis
- Löffler's syndrome
- sarcoidosis
- scleroderma

Rheumatic fever: criteria

Rheumatic fever develops following an immunological reaction to recent (2-6 weeks ago) *Streptococcus pyogenes* infection. **Diagnosis is based on evidence of recent streptococcal infection accompanied by:**

- 2 major criteria
- 1 major with 2 minor criteria

Evidence of recent streptococcal infection

- ASOT > 200iu/mL
- history of scarlet fever
- positive throat swab
- increase in DNase B titre.

Major criteria

- erythema marginatum
- Sydenham's chorea
- polyarthritis
- carditis (endo-, myo- or peri-)
- subcutaneous nodules

Minor criteria

- raised ESR or CRP
- pyrexia
- arthralgia (not if arthritis a major criteria)
- prolonged PR interval



© Image used on license from [DermNet NZ](https://www.dermnetnz.org/)



Erythema marginatum is seen in around 10% of children with rheumatic fever. It is rare in adults

External Link

[DermNet NZ](https://www.dermnetnz.org/)

Rheumatic fever

Supraventricular tachycardia

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.

Acute management

- vagal manoeuvres: e.g. Valsalva manoeuvre
- intravenous adenosine 6mg → 12mg → 12mg: contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes

- beta-blockers
- radio-frequency ablation

External Links

[Resuscitation Council UK](#)

2015 Peri-Arrest Arrhythmias

[Life in the Fast Lane](#)

Supraventricular tachycardia

Takayasu's arteritis

Takayasu's arteritis is a large vessel vasculitis. It typically causes occlusion of the aorta and questions commonly refer to an absent limb pulse. It is more common in females and Asian people

Features

- systemic features of a vasculitis e.g. malaise, headache
- unequal blood pressure in the upper limbs
- carotid bruit
- intermittent claudication
- aortic regurgitation (around 20%)



Angiography showing multiple stenoses in the branches of the aorta secondary to Takayasu's arteritis

Associations

- renal artery stenosis

Management

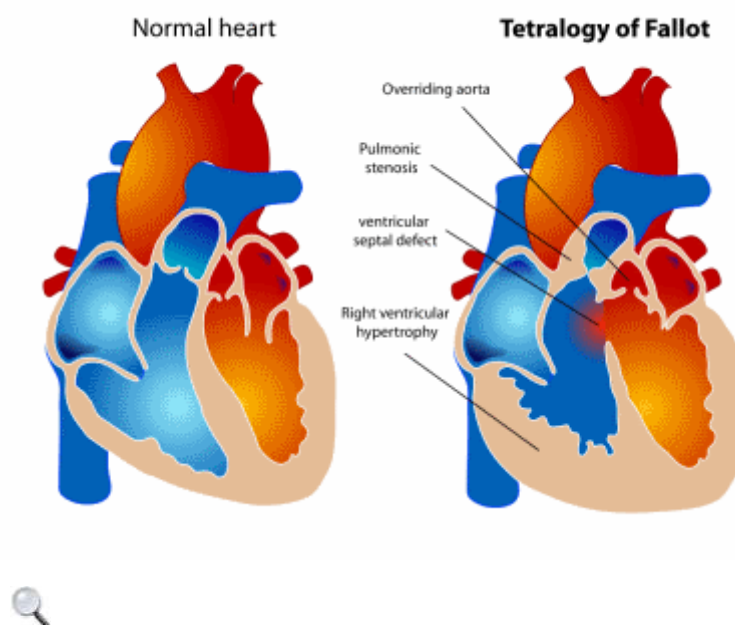
- steroids
-

Tetralogy of Fallot

Tetralogy of Fallot (TOF) is the most common cause of cyanotic congenital heart disease*. It typically presents at around 1-2 months, although may not be picked up until the baby is 6 months old

TOF is a result of anterior malalignment of the aortopulmonary septum. The four characteristic features are:

- ventricular septal defect (VSD)
- right ventricular hypertrophy
- right ventricular outflow tract obstruction, pulmonary stenosis
- overriding aorta



♦The severity of the right ventricular outflow tract obstruction determines the degree of cyanosis and clinical severity

Other features

- cyanosis
- causes a right-to-left shunt
- ejection systolic murmur due to pulmonary stenosis (the VSD doesn't usually cause a murmur)
- a right-sided aortic arch is seen in 25% of patients
- chest x-ray shows a 'boot-shaped' heart, ECG shows right ventricular hypertrophy

Management

- surgical repair is often undertaken in two parts
- cyanotic episodes may be helped by beta-blockers to reduce infundibular spasm

*however, at birth transposition of the great arteries is the more common lesion as patients with TOF generally present at around 1-2 months

Torsades de pointes

Torsades de pointes ('twisting of the points') is a rare arrhythmia associated with a long QT interval. It may deteriorate into ventricular fibrillation and hence lead to sudden death

Causes of long QT interval

- congenital: Jervell-Lange-Nielsen syndrome, Romano-Ward syndrome
- antiarrhythmics: amiodarone, sotalol, class 1a antiarrhythmic drugs
- tricyclic antidepressants
- antipsychotics
- chloroquine
- terfenadine
- erythromycin
- electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia
- myocarditis
- hypothermia
- subarachnoid haemorrhage

Management

- IV magnesium sulphate

External Links:

[Life in the Fast Lane](#)

Torsades de pointes.

Tricuspid regurgitation

Signs

- pan-systolic murmur
- giant V waves in JVP
- pulsatile hepatomegaly
- left parasternal heave

Causes

- right ventricular dilation
- pulmonary hypertension e.g. COPD
- rheumatic heart disease
- infective endocarditis (especially intravenous drug users)
- Ebstein's anomaly
- carcinoid syndrome

Ventricular septal defects

Ventricular septal defects (VSD) are the most common cause of congenital heart disease. They close spontaneously in around 50% of cases. Congenital VSDs are associated with chromosomal disorders (e.g. Down's syndrome, Edward's syndrome, Patau syndrome) and single gene disorders. Non-congenital causes include post myocardial infarction

Features

- classically a pan-systolic murmur which is louder in smaller defects

Complications

- aortic regurgitation*
- infective endocarditis
- Eisenmenger's complex
- right heart failure
- pulmonary hypertension: pregnancy is contraindicated in women with pulmonary hypertension as it carries a 30-50% risk of mortality

*aortic regurgitation is due to a poorly supported right coronary cusp resulting in cusp prolapse

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Ventricular tachycardia

Ventricular tachycardia (VT) is broad-complex tachycardia originating from a ventricular ectopic focus. It has the potential to precipitate ventricular fibrillation and hence requires urgent treatment.

There are two main types of VT:

- monomorphic VT: most commonly caused by myocardial infarction
- polymorphic VT: A subtype of polymorphic VT is torsades de pointes which is precipitated by prolongation of the QT interval. The causes of a long QT interval are listed below

Chapter: cardiology

• Causes of a prolonged QT interval

Congenital	Drugs	Other
• Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	☐ amiodarone, sotalol, class 1a antiarrhythmic drugs ☐ tricyclic antidepressants, fluoxetine ☐ chloroquine ☐ terfenadine ☐ erythromycin	☐ electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia ☐ acute myocardial infarction ☐ myocarditis ☐ hypothermia ☐ subarachnoid haemorrhage

Management

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure or rate > 150 beats/min) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks

Drug therapy

- amiodarone: ideally administered through a central line
- lidocaine: use with caution in severe left ventricular impairment
- procainamide

♦ *Verapamil should NOT be used in VT*

♦ If drug therapy fails

- electrophysiological study (EPS)
- implantable cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

External Links

[Resuscitation Council \(UK\)](#)

2010 Resuscitation Guidelines

Ventricular tachycardia: management

Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure, syncope) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks

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Peri-arrest arrhythmias

Warfarin

Warfarin is an oral anticoagulant which inhibits the reduction of vitamin K to its active hydroquinone form, which in turn acts as a cofactor in the carboxylation of clotting factor II, VII, IX and X (mnemonic = 1972) and protein C.

Indications:

- venous thromboembolism: target INR = 2.5, if recurrent 3.5
- atrial fibrillation, target INR = 2.5
- mechanical heart valves, target INR depends on the valve type and location. Mitral valves generally require a higher INR than aortic valves.

Patients on warfarin are monitored using the INR (international normalised ratio), the ratio of the prothrombin time for the patient over the normal prothrombin time. Warfarin has a long half-life and achieving a stable INR may take several days. There is a variety of loading regimes and computer software is now often used to alter the dose.

Factors that may potentiate warfarin:

- liver disease
- P450 enzyme inhibitors, e.g.: amiodarone, ciprofloxacin
- cranberry juice
- drugs which displace warfarin from plasma albumin, e.g. NSAIDs
- inhibit platelet function: NSAIDs

Side-effects:

- haemorrhage
- teratogenic, although can be used in breast-feeding mothers
- skin necrosis: when warfarin is first started biosynthesis of protein C is reduced. This results in a temporary procoagulant state after initially starting warfarin, normally avoided by concurrent heparin administration. Thrombosis may occur in venules leading to skin necrosis
- purple toes

External Links:

[BCSH](#)

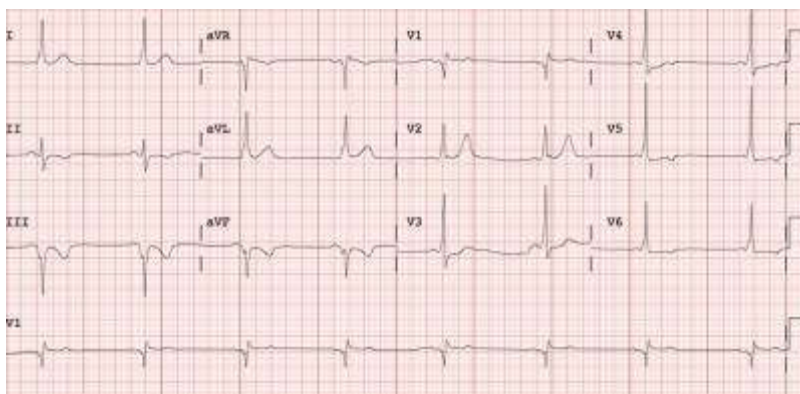
Guidelines for the management of patients on oral anticoagulants requiring dental surgery

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to a atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF

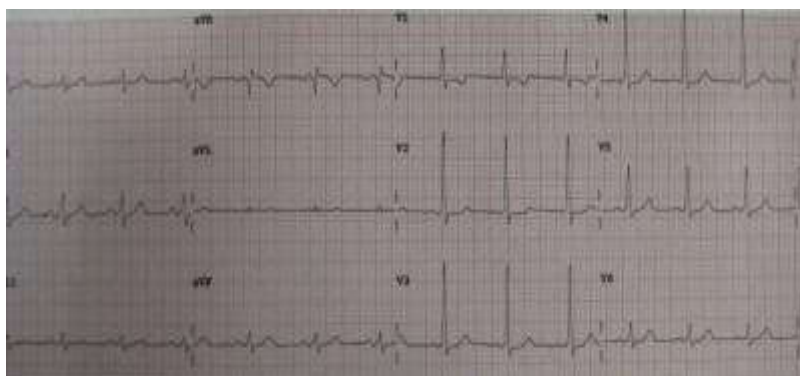
Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway*
- right axis deviation if left-sided accessory pathway*



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ECG showing short PR interval associated with a slurred upstroke (delta wave). Note the non-specific ST-T changes which are common in WPW and may be mistaken for ischaemia. The left axis deviation means that this is type B WPW, implying a right-sided pathway



© Image used on license from [Dr Smith, University of Minnesota](#)

Further example showing a characteristic delta wave

Differentiating between type A and type B**

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

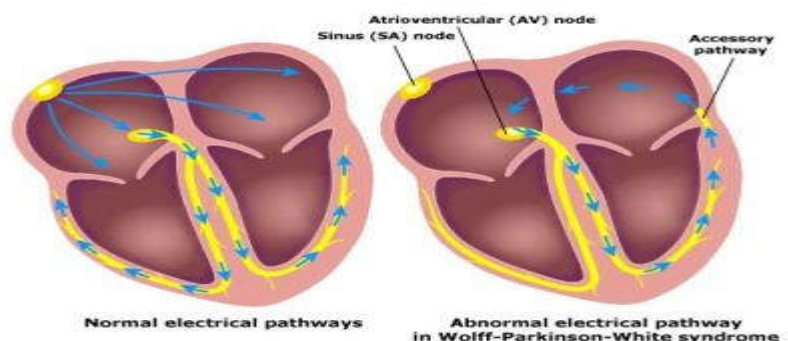


Image sourced from [Wikipedia](#)



Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide

*in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation

**there is a rare type C WPW, WPW in which the delta waves are upright in leads V1-V4 but negative in leads V5-V6

***sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation